

WEST Search History

DATE: Sunday, July 13, 2003

<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
side by side			result set
<i>DB=USPT; PLUR=YES; OP=ADJ</i>			
L13	L12 and (l1 or l2 or l3 or l4) not (l6 or l7)	9	L13
L12	l5.ti,ab,clm.	156	L12
L11	L10 same (l1 or l2 or l3 or l4)	19	L11
L10	clostrid\$	4558	L10
L9	L5 same l4	5	L9
L8	L5 same l3	5	L8
L7	L5 same l2	5	L7
L6	L5 same l1	16	L6
L5	botulin\$6 or botox	1119	L5
L4	hypocalcem\$ or hypercalcem\$	1572	L4
L3	hypothyroid\$ or hyperthyroid\$	1440	L3
L2	calcitonin	3315	L2
L1	thyroid or thyroxin	9105	L1

END OF SEARCH HISTORY

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Stedman's Dictionary

Define:

Stedman's Medical Dictionary 27th Edition

calcitonin (kal-si-to'nin)

A peptide hormone, of which eight forms in five species are known; composed of 32 amino acids and produced by the parathyroid, thyroid, and thymus glands; its action is opposite to that of parathyroid hormone in that c. increases deposition of calcium and phosphate in bone and lowers the level of calcium in the blood; its level in the blood is increased by glucagon and by Ca^{2+} and thus opposes postprandial hypercalcemia. SYN: thyrocalcitonin. [calci- + G. tonos, stretching, + -in]

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Stedman's Medical Dictionary 27th Edition

hyperthyroidism (hi-per-thi'royd-izm)

An abnormality of the thyroid gland in which secretion of thyroid hormone is usually increased and is no longer under regulatory control of hypothalamic-pituitary centers; characterized by a hypermetabolic state, usually with weight loss, tremulousness, elevated plasma levels of thyroxin and/or triiodothyronine, and sometimes exophthalmos; may progress to severe weakness, wasting, hyperpyrexia, and other manifestations of thyroid storm; often associated with exophthalmos (Graves disease). SEE ALSO: thyrotoxicosis. SYN: hyperthyrea, thyroidism (1), thyrointoxication. **hereditary h.** a rare inherited (autosomal dominant) disorder with constitutive stimulation of the thyrocytes. **iodine-induced h.** SYN: Jod-Basedow (Jod-Basedow) phenomenon. **masked h. h.** occurring without the usual manifestations, especially lack of hyperactivity and eye findings, often with hypoactivity, even somnolence. Manifestation can be limited to heart failure. **ophthalmic h.** SYN: Graves disease. **primary h. h.** due to a disorder originating within the thyroid gland, in contrast to one of pituitary origin; may be due to generalized overactivity of the gland, to a localized hyperactive nodule, or to circulating antibody, which stimulates the gland (long-acting thyroid stimulator). **secondary h. h.** due to stimulation of the thyroid gland by an excess of thyrotrophin secreted by the pituitary gland.

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File 155:MEDLINE(R) 1966-2001/Jul W2 (c)
format only 2001 Dialog Corporation

Set Items Description

Ref Items RT Index-term
E1 1 THYROID
E2 1 THYROIDCYTES
E3 90754 *THYROID
E4 0 1 THYROID ANTAGONISTS
E5 0 1 THYROID CANCER
E6 516 3 THYROID CARTILAGE
E7 21 THYROID CARTILAGE --ABNORMALITIES
--AB
E8 59 THYROID CARTILAGE -ANATOMY AND
HISTOLOGY AH
E9 4 THYROID CARTILAGE --BLOOD SUPPLY --
BS
E10 4 THYROID CARTILAGE --CHEMISTRY --CH
E11 3 THYROID CARTILAGE --CYTOLOGY --CY
E12 1 THYROID CARTILAGE --EMBRYOLOGY --
EM
E13 5 THYROID CARTILAGE --GROWTH AND
DEVELOPMENT --G
E14 46 THYROID CARTILAGE --INJURIES --IN
E15 2 THYROID CARTILAGE --INNERVATION --IR
E16 3 THYROID CARTILAGE --METABOLISM --ME
E17 69 THYROID CARTILAGE --PATHOLOGY --PA
E18 25 THYROID CARTILAGE --PHYSIOLOGY --PH
E19 8 THYROID CARTILAGE --
PHYSIOPATHOLOGY --PP
E20 1 THYROID CARTILAGE --RADIATION
EFFECTS --RE
E21 49 THYROID CARTILAGE --RADIOGRAPHY --
RA
E22 5 THYROID CARTILAGE --RADIONUCLIDE
IMAGING --RI
E23 271 THYROID CARTILAGE --SURGERY --SU
E24 6 THYROID CARTILAGE --TRANSPLANTATION
--TR
E25 3 THYROID CARTILAGE --
ULTRASONOGRAPHY --US
E26 4 THYROID CARTILAGE --
ULTRASTRUCTURE --UL
E27 494 5 THYROID CRISIS
E28 29 THYROID CRISIS --BLOOD --BL
E29 30 THYROID CRISIS --CHEMICALLY
INDUCED --CI
E30 77 THYROID CRISIS --COMPLICATIONS --
CO
E31 98 THYROID CRISIS --DIAGNOSIS --DI
E32 136 THYROID CRISIS --DRUG THERAPY --DT
E33 2 THYROID CRISIS --ENZYMOLGY --EN
E34 6 THYROID CRISIS --EPIDEMIOLOGY --EP
E35 123 THYROID CRISIS --ETIOLOGY --ET
E36 3 THYROID CRISIS --IMMUNOLOGY --IM
E37 13 THYROID CRISIS --METABOLISM --ME
E38 7 THYROID CRISIS --MORTALITY --MO
E39 12 THYROID CRISIS --NURSING --NU
E40 5 THYROID CRISIS --PATHOLOGY --PA
E41 36 THYROID CRISIS --PHYSIOPATHOLOGY --
PP
E42 35 THYROID CRISIS --PREVENTION AND
CONTROL --PC
E43 1 THYROID CRISIS --RADIOGRAPHY --RA
E44 3 THYROID CRISIS --RADIONUCLIDE
IMAGING --RI
E45 1 THYROID CRISIS --RADIOTHERAPY --RT
E46 12 THYROID CRISIS --SURGERY --SU
E47 140 THYROID CRISIS --THERAPY --TH
E48 9605 21 THYROID DISEASES

S1 90754 "THYROID"
S2 9605 "THYROID DISEASES"

Ref Items Type RT Index-term
R1 9605 21 *THYROID DISEASES
R2 9605 X DC=C19.874. (THYROID DISEASES)
R3 786 N 9 CRETINISM

R4 192 N 6 EUTHYROID SICK SYNDROMES
R5 11460 N 6 GOITER
R6 1512 N 2 GOITER, ENDEMIC
R7 2024 N 3 GOITER, NODULAR
R8 471 N 3 GOITER, SUBSTERNAL
R9 8883 N 15 GRAVES' DISEASE
R10 18660 N 6 HYPERTHYROIDISM
R11 276 N 3 HYPERTHYROXINEMIA
R12 19237 N 5 HYPOTHYROIDISM

S3 9605 DC="C19.874."
S4 786 "CRETINISM"
S5 192 "EUTHYROID SICK SYNDROMES"
S6 11460 "GOITER"
S7 1512 "GOITER, ENDEMIC"
S8 2024 "GOITER, NODULAR"
S9 471 "GOITER, SUBSTERNAL"
S10 8883 "GRAVES' DISEASE"
S11 18660 "HYPERTHYROIDISM"
S12 276 "HYPERTHYROXINEMIA"
S13 19237 "HYPOTHYROIDISM"
S14 111387 S1 OR (S2-S13)
S15 16877 CLOSTRIDIUM
S16 14 S14 AND S15
S17 49003 NEUROTOXIN OR TOXIN
S18 323 S14 AND S17

Ref Items RT Index-term
E1 34 BOTULINUM TOXIN TYPE E
E2 14 BOTULINUM TOXIN TYPE F
E3 3071 11 *BOTULINUM TOXINS
E4 568 BOTULINUM TOXINS --
ADMINISTRATION AND DOSAGE -
E5 251 BOTULINUM TOXINS --ADVERSE
EFFECTS --AE
E6 248 BOTULINUM TOXINS --ANALYSIS --AN
E7 34 BOTULINUM TOXINS --ANTAGONISTS
AND INHIBITORS
E8 168 BOTULINUM TOXINS --BIOSYNTHESIS
--BI
E9 64 BOTULINUM TOXINS --BLOOD --BL
E10 1 BOTULINUM TOXINS --CHEMICAL
SYNTHESIS --CS
E11 119 BOTULINUM TOXINS --CHEMISTRY --
CH
E12 30 BOTULINUM TOXINS --
CLASSIFICATION --CL

Ref Items Type RT Index-term
R1 3071 11 *BOTULINUM TOXINS
R2 3071 X DC=D24.185.926.123.179.
(BOTULINUM TOXINS)
R3 3071 X DC=D24.185.926.640.75.
(BOTULINUM TOXINS)
R4 114 X 1 BOTULIN
R5 0 X 1 CLOSTRIDIUM BOTULINUM TOXINS
R6 1785 R 10 BOTULISM
R7 628 R 108 CHOLINERGIC AGENTS
R8 1519 R 5 CLOSTRIDIUM BOTULINUM
R9 301 B 29 ANTI-DYSKINESIA AGENTS
R10 9006 B 16 BACTERIAL TOXINS
R11 7222 B 15 NEUROTOXINS
R12 585 N 4 BOTULINUM TOXIN TYPE A

S19 3071 "BOTULINUM TOXINS"
S20 16 S19 AND S14
S21 9605 "THYROID DISEASES"
S22 0 DELETE S21
S23 0 S3 AND S18 AND (PERFRINGENS)
S24 8 S21 AND S

16/6/1 10026619 99097345 PMID: 9878248
Genes for the CPE receptor (CPETR1) and the human homolog of
RVPI(CPETR2) are localized within the Williams-Beuren syndrome
deletion. Dec 15 1998

16/6/2 04722792 81133651 PMID: 6258596
Demonstration and characterization of partial glyceride specific lipases
in pig thyroid plasma membranes. Nov 28 1980

16/6/3 04661467 84263155 PMID: 6378767

Def regulation of the immune response to tetanus toxoid in
Has disease. Jul 1984

16/6/4 04201303 81164223 PMID: 7215238
Gas-forming suppurative thyroiditis. Mar 1981

16/6/5 02675218 77138183 PMID: 191247
Effects of concanavalin A and neuraminidase on cyclic AMP levels
and 14C-1-glucose oxidation in dog thyroid slices. Aug 1976

16/6/6 02255653 69254786 PMID: 4895160
[Various findings on the processes of natural detoxication of the body
during infectious processes] Nekotorye dannye o protsessakh
estesvennoi detoksikatsii organizma pri infektsionnykh protsessakh. Oct
1967

16/6/7 02092271 74150094 PMID: 4363284
Possibility of cancer diagnosis by detection of Clostridial antibodies. 1972

16/6/8 02044882 71067137 PMID: 4321500
Endemic goiter in Greece: some new epidemiologic studies. Feb 1971

16/6/9 01943289 73001076 PMID: 4560621
Treatment of malignant tumors with spores of Clostridium butyricum M
55. 3. Therapeutic experiments on metastasizing tumors of various
organs] Die Behandlung maligner Geschwulste mit sporen von
Clostridium butyricum M 55. 3. Therapieversuche an metastasierenden
Geschwulsten verschiedener Organe. 1972

16/6/10 01695705 67139798 PMID: 4960830
Action of phospholipase C on the thyroid. Abolition of the response to
thyroid-stimulating hormone. Apr 25 1967.

16/6/11 01512434 70106909 PMID: 4312983
Role of lecithin in the mechanism of TSH action. Apr 1970

16/6/12 01502036 68318489 PMID: 4298230
Effect of sphingomyelinase from Clostridium perfringens on the
metabolic activity and phospholipid composition of thyroid slices. Jul 1
1968

16/6/13 01496873 67221632 PMID: 4292226
The purification and properties of a thyroid-stimulating factor
isolated from Clostridium perfringens. Aug 25 1967

16/6/14 00882933 71059144 PMID: 5487765
Clostridium septicum infection of the thyroid gland. Sep 1970

16/7/1 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
10026619 99097345 PMID: 9878248

Genes for the CPE receptor (CPETR1) and the human homolog of RVP1 (CPETR2) are localized within the Williams-Beuren syndrome deletion.

Paperna T; Peoples R; Wang YK; Kaplan P; Francke U

Department of Genetics, Stanford University School of Medicine, Stanford, California, 94305, USA.

Genomics (UNITED STATES) Dec 15 1998, 54 (3) p453-9, ISSN 0888-7543 Journal Code: GEN Contract/Grant No.: HD01181, HD, NICHD; HD33505, HD, NICHD; HG00298, HG, NHGRI Languages: ENGLISH Document type: Journal Article Record type: Completed

Williams-Beuren syndrome (WBS) is a neurodevelopmental disorder affecting multiple systems. Haploinsufficiency of genes deleted in chromosomal region 7q11.23 is the likely cause for this syndrome. We now report the localization of the genes for the CPE-R (Clostridium perfringens enterotoxin receptor, CPETR1) and the human homolog of RVP1 (rat ventral prostate 1 protein, CPETR2), both previously mapped to 7q11, to the WBS critical region. A single nucleotide polymorphism (SNP) present in CPETR1 has been identified and was used to determine parental origin of the deleted allele in five informative families. The mouse homologs Cpetr1 and Cpetr2 were identified and mapped to the conserved syntenic region on mouse chromosome 5. Northern blot analysis of CPETR1 demonstrates tissue specificity, with expression in kidney, lung, thyroid, and gastrointestinal tissues. In mouse, Cpetr1 is expressed in the early embryo, appears to be developmentally upregulated during gestation, and is present in adult tissues. Our results suggest a role for CPE-R in internal organ development and function during pre- and postnatal life.

Copyright 1998 Academic Press. Record Date Created: 19990223

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04722792 81133651 PMID: 6258596

Demonstration and characterization of partial glyceride specific lipases in pig thyroid plasma membranes.

Igarashi Y; Kondo Y

Biochemical and biophysical research communications (UNITED STATES) Nov 28 1980, 97 (2) p766-71, ISSN 0006-291X Journal Code: 9Y8 Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19810413

16/7/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
04661467 84263155 PMID: 6378767

Defective regulation of the immune response to tetanus toxoid in Hashimoto's disease.

Fawcett J; Hutton C; McLachlan SM; Clark F; Rees Smith B

Immunology (ENGLAND) Jul 1984, 52 (3) p525-8, ISSN 0019-2805 Journal Code: GH7

Languages: ENGLISH Document type: Journal Article Record type: Completed

The humoral immune response to tetanus toxoid has been studied in patients with Hashimoto's disease. Although the magnitude of the response was similar to that observed in normal subjects, the Hashimoto patients demonstrated an inability to regulate their levels of tetanus toxoid antibody. This apparent defect in the control of antibody synthesis may be an important factor in both the initiation and perpetuation of autoimmune thyroid disease. Record Date Created: 19840829

16/7/4 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
04201303 81164223 PMID: 7215238

Gas-forming suppurative thyroiditis.

Michel RG; Hall DM; Woodard BH

Ear, nose, & throat journal (UNITED STATES) Mar 1981, 60 (3) p127-30, ISSN 0145-5613 Journal Code: EDF

Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19810613

16/7/5 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
02675218 77138183 PMID: 191247

18/6/1 11223191 21167934 PMID: 11267996

Mechanisms of P2 receptor-evoked DNA synthesis in thyroid FRTL-5 cells. May 2001

18/6/2 10931041 20494849 PMID: 11041451

The thyrotropin receptor is not involved in the activation of p42/p44 mitogen-activated protein kinases by thyrotropin preparations in Chinese hamster ovary cells expressing the human thyrotropin receptor. Sep 2000

18/6/3 10880922 20406492 PMID: 10951975

Altered expression of G proteins in thyroid gland adenomas obtained from hyperthyroid cats. Aug 2000

18/6/4 10709815 20327831 PMID: 10867750

Is autism a G-alpha protein defect reversible with natural vitamin A? Jun 2000

18/6/5 10648056 20322683 PMID: 10866314

Cancer gene therapy by thyroid hormone-mediated expression of toxin genes. Jun 15 2000

18/6/6 10607495 20252140 PMID: 10794166

Study of the olivocochlear neurons using two different tracers, fast blue and cholera toxin, in hypothyroid rats. Apr 2000

18/6/7 10557836 20202319 PMID: 10737891

Effects of concanavalin A and neuraminidase on cyclic AMP levels and 14C-1-glucose oxidation in dog thyroid slices.

Yamashita K; Aiyoshi Y; Oka H; Ogata E

Endocrinologia japonica (JAPAN) Aug 1976, 23 (4) p355-8, ISSN 0013-7219 Journal Code: EG5 Languages: ENGLISH Document type: Journal Article Record type: Completed

Treatment with concanavalin A at 100 micron/ml or higher concentrations significantly increased 14C-1-glucose oxidation in dog thyroid slices as reported in other tissues. This treatment exerted no effect on tissue cyclic AMP levels. Neuraminidase at the same concentrations also had similar effects on these parameters. Neither concanavalin A nor neuraminidase at the concentrations up to 100 micron/ml had the TSH effect on both tissue cyclic AMP and 14C-1-glucose oxidation. These results indicate that modification of carbohydrate moieties of glycoproteins on the cell surface may cause an increase in glucose metabolism without any critical effect on cyclic AMP system and in the process of TSH response. Record Date Created: 19770527

16/7/9 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
01943289 73001076 PMID: 4560621

Treatment of malignant tumors with spores of Clostridium butyricum M 55. 3. Therapeutic experiments on metastasizing tumors of various organs] Die Behandlung maligner Geschwulste mit sporen von Clostridium butyricum M 55. 3. Therapieversuche an metastasierenden Geschwulsten verschiedener Organe.

Kretschmer H; Glaser A; Grasser A

Archiv für Geschwulstforschung (GERMANY, EAST) 1972, 39 (4) p315-21, ISSN 0003-911X Journal Code: 746 Languages: GERMAN Document type: Clinical Trial; Journal Article Record type: Completed Record Date Created: 19721110

16/5/10 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
01695705 67139798 PMID: 4960830

Action of phospholipase C on the thyroid. Abolition of the response to thyroid-stimulating hormone.

Macchia V; Pastan I

Journal of biological chemistry (UNITED STATES) Apr 25 1967, 242 (8) p1864-9, ISSN 0021-9258 Journal Code: HIV Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19670706

Tags: Animal; In Vitro

Descriptors: Phospholipases--pharmacology--PD; *Thyroid Gland--drug effects--DE; *Thyrotropin--pharmacology--PD; Acetylcholine--pharmacology--PD; Clostridium--enzymology--EN; Dogs; Edetic Acid--pharmacology--PD; Glucose--metabolism--ME; Neuraminidase--metabolism--ME

CAS Registry No.: 50-99-7 (Glucose); 51-84-3 (Acetylcholine); 60-00-4 (Edetic Acid); 9002-71-5

(Thyrotropin)

Enzyme No.: EC 3.1.- (Phospholipases); EC 3.2.1.18 (Neuraminidase)

16/5/13 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
01496873 67221632 PMID: 4292226

The purification and properties of a thyroid-stimulating factor isolated from Clostridium perfringens.

Macchia V; Bates RW; Pastan I

Journal of biological chemistry (UNITED STATES) Aug 25 1967, 242 (16) p3726-30, ISSN 0021-9258 Journal Code: HIV Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19671022

Tags: Animal; In Vitro

Descriptors: Bacterial Proteins--analysis--AN; *Bacterial Proteins--pharmacology--PD; *Clostridium perfringens--analysis--AN; *Thyroid Gland--metabolism--ME; Carbon Dioxide; Carbon Isotopes; Cattle; Chromatography, Gel; Dogs; Glucose--metabolism--ME; Molecular Weight; Peptide Hydrolases; Phosphates--metabolism--ME; Phospholipids--biosynthesis--BI; Thyrotropin; Trypsin

CAS Registry No.: 0 (Bacterial Proteins); 0 (Carbon Isotopes); 0 (Phosphates); 0 (Phospholipids); 124-38-9 (Carbon Dioxide); 50-99-7 (Glucose); 9002-71-5 (Thyrotropin)

Enzyme No.: EC 3.4 (Peptide Hydrolases); EC 3.4.21.4 (Trypsin)

Extracellular ATP-mediated phospholipase A(2) activation in rat thyroid FRTL-5 cells: regulation by a G(i)/G(o) protein, Ca(2+), and mitogen-activated protein kinase. May 2000

18/6/8 10516902 20163705 PMID: 10701770

Effect of endotoxin challenge on hepatic 5'-deiodinase activity in cattle. Jan 2000

18/6/9 10513968 20115224 PMID: 10648115

Adjuvant effects of cholera toxin b subunit on immune response to recombinant thyrotropin receptor in mice. Feb 2000

18/6/10 10359010 20000371 PMID: 10532571

What is the role of botulinum toxin in the treatment of dysthyroid strabismus? Oct 1999

18/6/11 10358931 20000569 PMID: 10532769

Strabismus surgery among aged medicare beneficiaries. Dec 1997

18/6/12 10341274 99328094 PMID: 10401667

An adenosine receptor agonist-induced modulation of TSH-dependent cell growth in FRTL-5 thyroid cells mediated by inhibitory G protein, Gi. Apr 1999

18/6/13 10336103 99262181 PMID: 10329469

Sphingosylphosphorylcholine activates Gq, Gi-2, and Gi-3 in thyroid FRTL-5 cells: implications for the activation of calcium fluxes and Na+-H+ exchange. May 1999

18/6/14 10335016 99261834 PMID: 10329948

Thyroid hormone induces activation of mitogen-activated protein kinase in cultured cells. May 1999

18/6/15 10291747 97423434 PMID: 9277376

Effects of triiodothyronine administration on the adenylyl cyclase system in brown adipose tissue of rat. Aug 1997

18/6/16 10240117 99371424 PMID: 10443824

The posterior thyroplasty window: anatomical considerations. Aug 1999

18/6/17 10180055 99302523 PMID: 10374293

[Systemic manifestations of myasthenia gravis and its putative pathogenesis] Jun 1997

18/6/18 10076868 99196145 PMID: 10098509

Thyrotropin regulates c-Jun N-terminal kinase (JNK) activity through two distinct signal pathways in human thyroid cells. Apr 1999

18/6/19 10058220 99165190 PMID: 10067867

Regulation and transfer of a murine model of thyrotropin receptor antibody mediated Graves' disease. Mar 1999

18/6/20 10021711 99082623 PMID: 9865104
Bilateral laryngeal movement disorder and synkinesia: value of botulism toxin. Apropos of a case] Trouble de la mobilité laryngée bilatérale et synkinesies: intérêt de la toxine botulique. A propos d'un cas. 1998

18/6/21 09880412 98421840 PMID: 9751220
Cyclic AMP impairs the PRL stimulation of iodide uptake into mouse mammary tissues. Oct 1998

18/6/22 09828189 98361458 PMID: 9697993
Characterization of the murine immune response to the murine TSH receptor ectodomain: induction of hypothyroidism and TSH receptor antibodies. Jul 1998

18/6/23 09825762 98378683 PMID: 9713061
Botulinum toxin A treatment of overactive corrugator supercilii in thyroid eye disease. May 1998

18/6/24 09783338 98283956 PMID: 9618427
Effect of antithyroid drugs on hydroxyl radical formation and alpha-1-proteinase inhibitor inactivation by neutrophils: therapeutic implications. Jun 1998

18/6/25 09700286 98184392 PMID: 9525480
Protein tyrosine phosphorylation and calcium signaling in thyroid FRTL-5 cells. May 1998

18/6/26 09674340 98070720 PMID: 9405207
Loss of biological activity due to Glu->Arg mutation at residue 11 of the B subunit of cholera toxin. Nov 1997

18/6/27 09589335 97462684 PMID: 9322911
Sphingosine 1-phosphate mobilizes sequestered calcium, activates calcium entry, and stimulates deoxyribonucleic acid synthesis in thyroid FRTL-5 cells. Oct 1997

18/6/28 09579710 97424751 PMID: 9278864
Somatostatin blocks the potentiation of TRH-induced TSH secretion from perfused pituitary fragments and the change in intracellular calcium concentrations from dispersed pituitary cells elicited by prepro-TRH (PS4) or by tri-iodothyronine. Aug 1997

18/6/29 09568457 97405869 PMID: 9260913
The phosphatase inhibitor okadaic acid stimulates the TSH-induced G1-S phase transition in thyroid cells. Aug 1 1997

18/6/30 09470883 98025576 PMID: 9376224
Sodium saccharin inhibits adenylyl cyclase activity in non-taste cells. Sep 1997

18/6/31 09469678 97375443 PMID: 9231760
Thyroid-specific expression of cholera toxin A1 subunit causes thyroid hyperplasia and hyperthyroidism in transgenic mice. Aug 1997

18/6/32 09463997 97254493 PMID: 9099903
Multiple G-protein coupling of the dog thyrotropin receptor. Mar 14 1997

18/6/33 09459986 97240583 PMID: 9124507
Sphingosine 1-phosphate stimulates Na+/H+ exchange in thyroid FRTL-5 cells. Mar 1997

18/6/34 09455989 97131950 PMID: 8977407
Sphingosine 1-phosphate stimulates hydrogen peroxide generation through activation of phospholipase C-Ca2+ system in FRTL-5 thyroid cells: possible involvement of guanosine triphosphate-binding proteins in the lipid signaling. Jan 1997

18/6/35 09428059 98018536 PMID: 9380360
Chemodenervation in treatment of upper eyelid retraction. 1997

18/6/36 09408392 98018742 PMID: 9376076
TCR beta usage of TSH receptor-specific CD4+ T cells in Graves' disease patients and healthy humans. Oct 1997

18/6/37 09372529 97366554 PMID: 9223384
N6-isopentenyladenosine affects cAMP-dependent microfilament organization in FRTL-5 thyroid cells. Jul 10 1997

18/6/38 09342718 97329812 PMID: 9186271
Assessment of thyroid growth stimulating activity of immunoglobulins from patients with autoimmune thyroid disease by cytokinesis arrest assay. May 1997

18/6/39 09338863 97324798 PMID: 9180903
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Botulinum toxin for the treatment of dysthyroid ocular myopathy. Apr 1986

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Botulinum toxin for the treatment of dysthyroid ocular myopathy. Apr 1986

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Botulinum toxin for the treatment of dysthyroid ocular myopathy. Apr 1986

Tags: Case Report; Female; Human; Male; Support, Non-U.S. Gov't

Descriptors: Botulinum Toxins--therapeutic use--TU;

*Ophthalmoplegia--drug therapy--DT; *Thyroid Diseases--

complications--CO; Adult; Aged; Amblyopia--drug therapy--DT;

Amblyopia--etiology--ET; Botulinum Toxins--adverse effects--AE;

Middle Age; Ophthalmoplegia--complications--CO; Ophthalmoplegia--

etiology--ET

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What is the role of botulinum toxin in the treatment of dysthyroid strabismus?

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Journal of AAPOS(UNITED STATES) Oct 1999, 3 (5) p272-4, ISSN 1091-8531 Journal

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BACKGROUND: Botulinum toxin A has been used in the treatment of dysthyroid strabismus

primarily as a temporary measure during the active phase of the disease. We report on our

experience with 65 patients. METHOD: We review the records of 65 patients with dysthyroid

strabismus who were treated with botulinum toxin A at Moorfields Eye Hospital between 1984 and

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1996. CONCLUSIONS: Patients with a short duration of relatively mild dysthyroid strabismus have a chance of long-term benefit with botulinum toxin A. There is little use for botulinum toxin A in cases of severe dysthyroid disease. Record Date Created: 19991123

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Strabismus surgery among aged medicare beneficiaries.

Repka MX

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Inhibition of thyrotropin-stimulated adenosine 3',5'-monophosphate formation in rat thyroid cells by an adenosine analog. Evidence that the inhibition is mediated by the putative inhibitory guanine nucleotide regulatory protein. 1986

18/6/185 05999964 87016915 PMID: 3020540

Phospholipase A2 and phospholipase C are activated by distinct GTP-binding proteins in response to alpha 1-adrenergic stimulation in FRTL5 thyroid cells. Oct 1986

18/6/186 05998196 87128012 PMID: 3028381

Role of pertussis toxin sensitive G proteins in the alpha 1 adrenergic receptor but not in the thyrotropin receptor mediated activation of membrane phospholipases and iodide fluxes in FRTL-5 thyroid cells. Dec 30 1986

18/6/187 05998164 87126855 PMID: 2434027

Alpha 1-adrenergic regulation of TSH-stimulated cyclic AMP accumulation in rat thyroid cells. Feb 15 1987

18/6/188 05993703 86274409 PMID: 2426089

Evidence that adenosine 3',5'-monophosphate mediates stimulation of thyroid growth in FRTL5 cells. Aug 1986

18/6/189 05990130 86079192 PMID: 3000588

Stimulation of cell proliferation and inhibition of differentiation expression by tumor-promoting phorbol esters in dog thyroid cells in primary culture. Feb 1986

18/6/190 05986692 86030260 PMID: 2996887

Antagonistic effects of thyrotropin and epidermal growth factor on thyroglobulin mRNA level in cultured thyroid cells. Oct 15 1985

18/6/191 05985339 85286022 PMID: 2411613

Studies on the mechanism of desensitization of the cyclic AMP response to TSH stimulation in a cloned rat thyroid cell line. Aug 1985

18/6/192 05983357 85204900 PMID: 2987064

Thyrotropin modulates EGF receptor function in porcine thyroid follicle cells. Apr 1985

18/6/193 05981918 85131002 PMID: 2982825

Fat cell adenylate cyclase system. Enhanced inhibition by adenosine and GTP in the hypothyroid rat. Feb 25 1985

18/6/194 05948424 88297495 PMID: 2841216

The adenylate cyclase system and calcitonin secretion from perfused dog thyroid lobes. May 1988

18/6/195 05940674 89338240 PMID: 2547582

Insulin-like growth factor-I potentiates thyrotropin stimulation of adenylate cyclase in FRTL-5 cells. Sep 1989

18/6/196 05925481 89062561 PMID: 2848590

Homologous and heterologous beta-adrenergic desensitization in hepatocytes. Additivity and effect of pertussis toxin. Dec 9 1988

18/6/197 05911374 87294932 PMID: 3039775

Role of the adenylate cyclase-cAMP system on TSH-stimulated thyroid cell growth. 1987

18/6/198 05907845 88082851 PMID: 3691532

Iodide-induced inhibition of adenylate cyclase activity in horse and dog thyroid. Dec 30 1987

18/6/199 05900120 87247650 PMID: 3036626

Regulation of calcium fluxes in the thyroid. May 1987

18/6/200 05896398 87162673 PMID: 3030831

Adenylate cyclase system responsive to thyroid stimulating hormone (TSH) of porcine thyroid cells in primary monolayer cultures. Potential effect of forskolin on TSH-mediated adenylate cyclase stimulation] Jan 20 1987

OBJECTIVES: The purpose of this study was to investigate the incidence of strabismus surgery among aged patients in the United States. **METHODS:** The Medicare Part B claims experience (physician professional fee billing) for 1995 was reviewed for the number of times each strabismus surgical procedure recognized in Physicians' Current Procedural Terminology (CPT) was performed. To determine the indications for the procedures that were performed, a 5% sample of claims was reviewed for the pertinent International Classification of Diseases, Ninth Revision, Clinical Modification, diagnostic codes. **RESULTS:** There were 27 million aged Medicare beneficiaries eligible for Part B benefits in 1995 in a fee-for-service setting. During that year physicians reported 9497 strabismus physician services. These represented 6585 separate procedures (CPT codes 67311 to 67343) and 277 botulinum toxin (Botox) injections for strabismus (CPT 67345) performed during 1995. Sixty-nine percent of the surgical procedures were for horizontal correction and 28% were for vertical correction. Adjustable sutures were used for only 1240 cases (1.9%). The add-on procedural code for reoperation surgery or surgery in the presence of restriction of the extraocular muscles was used in just 930 cases (14%). The most common diagnosis for horizontal surgery was exotropia. Paralytic strabismus and thyroid disease were identified for 17% of cases. Three percent of the diagnoses were inappropriate for the procedures performed and may have been reported in error. **CONCLUSIONS:** These data confirm a very low incidence of strabismus surgical procedures (2/10,000) and injections (1/100,000) among aged Medicare beneficiaries. The strabismus surgery was most often performed to repair a horizontal deviation. The adjustable suture technique was used infrequently. These data may be extrapolated into the future to aid in determining the strabismus services that will be needed early in the next century. Record Date Created: 19991102

18/7/20 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 10021711 99082623 PMID: 9865104

Bilateral laryngeal movement disorder and synkinesia: value of botulinum toxin. Apropos of a case] Trouble de la mobilité laryngée bilatérale et syncinésies: intérêt de la toxine botulique. A propos d'un cas.

Marie JP; Navarre I; Leroisey Y; Magnier P; Dehesdin D; Andrieu Guitrancourt J
C.H.U. Rouen, Service d'ORL et Chirurgie Cervico-Faciale, France.

Revue de laryngologie - otologie - rhinologie (FRANCE) 1998, 119 (4) p261-4, ISSN 0035-1334 Journal Code: SDD

Languages: FRENCH Document type: Journal Article Record type: Completed

Several years after a subtotal thyroidectomy complicated by bilateral vocal cord paralysis, the patient presented with progressive dyspnea due to laryngeal synkinesia. The impairment of the ventilation status, in spite of laser arytenoidectomy, followed by contralateral posterior transverse cordotomy, suggested a botulinum toxin injection in the intrinsic adductor laryngeal muscles. The rapid improvement in ventilation without phonatory impairment is discussed in the following report. Record Date Created: 19990212

18/7/23 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09825762 98378683 PMID: 9713061

Botulinum toxin A treatment of overactive corrugator supercilii in thyroid eye disease.

Oliver JM

Western Eye Hospital, London.

British journal of ophthalmology (ENGLAND) May 1998, 82 (5) p528-33, ISSN 0007-1161

Journal Code: AZK Languages: ENGLISH Document type: Clinical Trial; Journal Article

Record type: Completed

BACKGROUND/AIM: Patients with thyroid eye disease with upper eyelid retraction often develop overaction of the accessory muscles of eyelid closure, the glabellar muscles corrugator supercilii and procerus. The resultant glabellar furrowing (frown lines) contributes to the typical thyroid facies. The aim of this study was to evaluate the use of botulinum toxin A reversible chemodenervation of the glabellar muscles as adjunctive treatment in the rehabilitation of patients with thyroid eye disease. **METHODS:** 14 patients (13 females) ages 39-76 years (mean 52) with inactive thyroid eye disease and associated medial eyebrow ptosis and prominent glabellar frown lines were recruited. All patients had a history of upper eyelid retraction. Each patient was treated with a single botulinum toxin injection (Dysport 0.2 ml, 40 units) into each corrugator supercilii and sometimes procerus muscles as an outpatient procedure. The effectiveness and acceptability of the treatment was assessed clinically and from a patient questionnaire. **RESULTS:** The injections were tolerated by 13/14 (93%) patients. There was resultant flattening of the glabellar region and improvement of medial eyebrow contour in all patients, with onset of paralysis within 1 week. All patients reported a subjective improvement in appearance. Side effects included one patient (7%) with reversible partial ptosis. The beneficial effect lasted 4-6 months, with a gradual return of function. Repeat treatment was indicated where there was persistent upper eyelid retraction and protractor overaction. **CONCLUSION:** Botulinum toxin A chemodenervation of the glabellar muscles in these patients was effective and acceptable. Chemodenervation should be considered in the rehabilitation of patients with thyroid eye disease where there is upper eyelid retraction and overacting protractors resulting in a thyroid frown. Once the eyelid retraction has been successfully treated by surgery, the need for further glabella muscle chemodenervation is considerably reduced. Record Date Created: 19980827

18/7/24 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09783338 98283956 PMID: 9618427

Effect of antithyroid drugs on hydroxyl radical formation and alpha-1-proteinase inhibitor inactivation by neutrophils: therapeutic implications.

Ross AD; Dey I; Janes N; Israel Y

Department of Pharmacology, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada M5S 1A8.

Journal of pharmacology and experimental therapeutics (UNITED STATES) Jun 1998, 285 (3) p1233-8, ISSN 0022-3565 Journal Code: JP3 Contract/Grant No.: 5P50 AA07186, AA, NIAAA; R01 AA10967, AA, NIAAA; R37-AA10967, AA, NIAAA

Languages: ENGLISH Document type: Journal Article Record type: Completed

The release of proteolytic enzymes and generation of strong oxidants such as the hydroxyl radical by activated neutrophils has been proposed to play an important role in mediating toxin-induced liver injury. The antithyroid drug propylthiouracil protects against liver injury induced by many hepatotoxic agents and markedly reduces mortality in patients with alcoholic liver disease. However, the mechanism(s) by which propylthiouracil protects against liver injury is not well understood. The present studies investigate the effect of antithyroid drugs on proteolytic enzyme activity and on hydroxyl radical generation from activated neutrophils. In the presence of hydrogen peroxide and chloride, neutrophil myeloperoxidase, an enzyme from the same gene superfamily as thyroid peroxidase, generates hypochlorous acid which inactivates alpha-1-proteinase inhibitor (A1PI) present in serum. This inactivation allows neutrophil-released proteolytic enzymes to attack cells. In the present study myeloperoxidase activity was inhibited fully at therapeutic concentrations by antithyroid drugs (propylthiouracil and methimazole). Antithyroid drugs fully prevented hypochlorous acid formation, and prevented neutrophil-mediated inactivation of A1PI, with concomitant blockage of proteolytic activity. Conversely, generation of both superoxide and hydroxyl radicals by activated neutrophils was unaffected by propylthiouracil. The production of these oxygen radicals was fully inhibited by the NADPH oxidase inhibitor diphenylene iodonium chloride, however. These studies indicate that antithyroid drugs are unlikely to prevent cell injury by inhibiting hydroxyl radical generation or by scavenging hydroxyl radicals, but are likely to exert their hepatoprotective anti-inflammatory action by inhibiting neutrophil myeloperoxidase, an enzyme akin to thyroid peroxidase. Record Date Created: 19980702

18/7/25 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09700286 98184392 PMID: 9525480

Protein tyrosine phosphorylation and calcium signaling in thyroid FRTL-5 cells.

Tomquist K; Dugue B; Ekoski E

Department of Biosciences, University of Helsinki, Finland.

Journal of cellular physiology (UNITED STATES) May 1998, 175 (2) p211-9, ISSN 0021-9541 Journal Code: HNB Languages: ENGLISH Document type: Journal Article Record type: Completed

We examined the importance of tyrosine kinase(s) on the ATP-evoked Ca²⁺ entry and DNA synthesis of thyroid FRTL-5 cells. ATP rapidly and transiently tyrosine phosphorylated a 72-kDa protein(s). This phosphorylation was abolished by pertussis toxin and by the tyrosine kinase inhibitor genistein, and was dependent on Ca²⁺ entry. Pretreatment of the cells with genistein did not affect the release of sequestered Ca²⁺, but the capacitative Ca²⁺ or Ba²⁺ entry evoked by ATP or thapsigargin was attenuated. Pretreatment of the cells with orthovanadate enhanced the increase in intracellular free Ca²⁺ ([Ca²⁺]_i), whereas the Ba²⁺ entry was not increased. Phorbol 12-myristate 13-acetate (PMA) phosphorylated the same protein(s) as did ATP. Genistein inhibited the ATP-evoked phosphorylation of MAP kinase and attenuated both the ATP- and the PMA-evoked DNA synthesis. However, genistein did not inhibit the ATP-evoked expression of c-fos. Furthermore, genistein enhanced the ATP-evoked release of arachidonic acid. Thus, ATP activates a tyrosine kinase via a Ca²⁺-dependent mechanism. A genistein-sensitive mechanism participates, in part, in the ATP-evoked activation of DNA synthesis. Genistein inhibits only modestly capacitative Ca²⁺ entry in FRTL-5 cells. Record Date Created: 19980416

18/7/26 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09674340 98070720 PMID: 9405207

Loss of biological activity due to Glu->Arg mutation at residue 11 of the B subunit of cholera toxin.

Yamaoka J; Yamasaki S; Kurazono H; Imamura S; Noda M; Miyai K; Takeda Y

Faculty of Medicine, Kyoto University, Kyoto, Sakyo-ku, 606-01, Japan.

Microbial pathogenesis (ENGLAND) Nov 1997, 23 (5) p297-302, ISSN 0882-4010

Journal Code: MIC

Languages: ENGLISH Document type: Journal Article Record type: Completed

Since it has been reported that a single amino acid mutation of Gly->Arg in the CAGYC region of the beta chain of human thyroid stimulating hormone (hTSH) was responsible for congenital isolated TSH deficiency, and that the same amino acid substitution in this site of hTSH and human chorionic gonadotropin (hCG) introduced by site-directed mutagenesis resulted in loss of activity, the authors studied the role of glutamic acid at position 11 (Glu-11) from the N-terminus of the B subunit of cholera toxin (CT), which corresponds to the glycine in the CAGYC region of the beta chain of hTSH and hCG. A mutant CT constructed by site-directed mutagenesis in which Glu-11 was replaced by Arg (CT-E11R) did not induce either morphological changes or accumulation of cytosolic cyclic AMP in Chinese hamster ovary cells, although it formed the holotoxin AB₅, retained the ability to bind to GM1-ganglioside and showed ADP-ribosyltransferase activity. Weak assembly of the B subunits in mutant CT-E11R demonstrated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis under non-heating conditions might explain the loss of biological activity. Copyright 1997 Academic Press Limited. Record Date Created: 19980317

18/7/31 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09469678 97375443 PMID: 9231760

Thyroid-specific expression of cholera toxin A1 subunit causes thyroid hyperplasia and hyperthyroidism in transgenic mice.

Zeiger MA; Saji M; Gusev Y; Westra WH; Takiyama Y; Dooley WC; Kohn LD; Levine MA

Department of Surgery, Johns Hopkins Medical Institutions, Baltimore, Maryland 21205, USA.

Endocrinology (UNITED STATES) Aug 1997, 138 (8) p3133-40, ISSN 0013-7227

Journal Code: EGZ Contract/Grant No.: RO-1 DK34281, DK, NIDDK Languages: ENGLISH

Document type: Journal Article Record type: Completed

Thyroid cell growth and function are regulated by hormones and growth factors binding to cell surface receptors that are coupled via G proteins, Gs and Gq, to the adenylyl cyclase and phospholipase C signal transduction systems, respectively. Activating mutations of the TSH receptor and G alpha s have been documented in subsets of thyroid neoplasms. To test the oncogenic potential of activated G alpha s in transgenic mice, we used the cholera toxin A1 subunit that constitutively activates G alpha s and used the rat thyroglobulin gene promoter for

targeting this transgene (TGCT) to thyroid follicular cells. Three (M1358, F1358, and F1286) of six founders identified were able to transmit the transgene to their offspring and thyroid glands from these mice contained elevated levels of cAMP. Concentrations of serum thyroxine were elevated as early as 2 months of age (M1392 and F1286). F1358 mice were euthyroid until 8 months of age, at which time they developed hyperthyroidism. All three TGCT lines developed thyroid hyperplasia independent of their thyroxine levels. DNA image analysis of thyroid follicular cells from both the hyper and euthyroid mice showed that DNA index and "S+G2/M" phase were increased compared with normal, changes similar to that seen in poor prognosis human carcinomas. These data suggest that the G alpha s-adenylyl cyclase-cAMP pathway has an important role in thyroid hyperplasia and the transgenic mouse models reported herein will allow further examination of the role of this pathway in thyroid oncogenesis. Record Date Created: 19970815

18/7/38 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09342718 97329812 PMID: 9186271

Assessment of thyroid growth stimulating activity of immunoglobulins from patients with autoimmune thyroid disease by cytokinesis arrest assay.

Miyamoto S; Kasagi K; Alam MS; Misaki T; Iida Y; Konishi J

Department of Nuclear Medicine, Faculty of Medicine, Kyoto University, Japan.

European journal of endocrinology (ENGLAND) May 1997, 136 (5) p499-507, ISSN

0804-4643 Journal Code: BXU Languages: ENGLISH Document type: Journal Article

Record type: Completed

OBJECTIVE: To develop a novel bioassay for the assessment of thyroid cell growth stimulating activity using cytochalasin B (CB) and to test immunoglobulins (IgGs) from patients with autoimmune thyroid diseases. **DESIGN:** The assay is based on the principle that growing cells during incubation with CB show an increased number of nuclei in a cell (N/C index), since CB, at appropriate concentrations, is known to inhibit cytoplasmic cleavage without affecting nuclear mitosis. The N/C index represents potential DNA production while cells are incubated with CB. **METHODS:** FRTL-5 thyroid cells were incubated with various thyroid stimulators in TSH-free medium containing 2 mg/l CB for 3 days. After the incubation, the cells were harvested in trypsin/EDTA to obtain single cell suspension, fixed, dropped onto a glass slide, stained and observed under a microscope to determine the N/C index. **RESULTS:** Bovine TSH at $10(-3)$ -1.0 U/l, forskolin at $1 \times 10(-7)$ - $10(-5)$ mol/l, cholera toxin at $10 \times 10(-5)$ - $10(-3)$ mg/l, or (Bu)₂cAMP at $1 \times 10(-5)$ - $10(-3)$ mol/l increased the N/C index up to approximately 2.0 in a dose-dependent manner. IgGs not only from 27 patients with untreated goitrous Graves' disease but also from 14 patients with goitrous Hashimoto's thyroiditis elicited an increase in the N/C index, which exceeded the mean + 2 S.D. of the values for 17 normal subjects (mean +/- S.D., 1.063 +/- 0.014). Four patients with primary myxedema displayed a normal N/C index. In Graves' disease, the N/C index did not correlate significantly with thyroid stimulating antibodies (TSAb) activities but did correlate significantly with estimated goiter size ($P < 0.05$). IgGs containing blocking-type TSH-receptor antibodies inhibited the TSH- or Graves IgG-stimulated increase in N/C index almost completely, but did not influence the stimulatory effect of IgG from two patients with Hashimoto's thyroiditis. **CONCLUSIONS:** We have developed a sensitive and simple assay for thyroid growth stimulating activity by using CB, and found that all tested patients with goitrous Graves' disease and goitrous Hashimoto's thyroiditis have thyroid growth stimulating immunoglobulins whose activity does not correlate with TSAb. Record Date Created: 19970710

18/7/45 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09159599 97154483 PMID: 9001201

Thyroid hormones as neurotransmitters.

Dratman MB; Gordon JT

Department of Medicine, MCP Hahnemann School of Medicine, Allegheny University, and Medical Research Service, Veterans Affairs Medical Center, Philadelphia, Pennsylvania 19104, USA.

Thyroid (UNITED STATES) Dec 1996, 6 (6) p639-47, ISSN 1050-7256 Journal Code: BJW Contract/Grant No.: 45252, PHS Languages: ENGLISH Document type: Journal Article; Review; Review, Tutorial Record type: Completed

During brain development, before the apparatus of neurotransmission has been set into place, many neurotransmitters act as growth regulators. In adult brain, their role in neurotransmission comes to the fore but neuronal plasticity and other growth-related processes are their continuing responsibility. This has been clearly demonstrated for catecholamines. Previous as well as recent evidence now indicates that thyroid hormones may participate in the developing and adult brain through similar mechanisms. Immunohistochemical mapping of brain triiodothyronine (antibody specificity established by numerous appropriate tests) demonstrated that the hormone was concentrated in both noradrenergic centers and noradrenergic projection sites. In the centers (locus coeruleus and lateral tegmental system) triiodothyronine staining, like that of tyrosine hydroxylase, was heavily concentrated in cytosol and cell processes. By contrast, in noradrenergic targets, label was most prominent in cell nuclei. Combined biochemical and morphologic data allows a construct of thyroid hormone circuitry to unfold: The locus coeruleus is conveniently located just beneath the ependyma of the 4th ventricle. Thyroxine, entering the brain via the choroid plexus, is preferentially delivered to subependymal brain structures. High concentrations of locus coeruleus norepinephrine promote active conversion of thyroxine to triiodothyronine, leading to the preeminence of the locus coeruleus as a site of triiodothyronine concentration. Results of treatment with the locus coeruleus neurotoxin DSP-4 established that axonal transport accounts for delivery of both triiodothyronine and norepinephrine from locus coeruleus to noradrenergic terminal fields. The apparatus for transduction of thyronergic and noradrenergic signals at both membrane and nuclear sites resides in the postsynaptic target cells. Upon internalization of hormone in post-synaptic target cells, genomic effects of triiodothyronine, norepinephrine, and/or their second messengers are possible and expected. The evidence establishes a direct morphologic connection between central thyronergic and noradrenergic systems, supporting earlier proposals that triiodothyronine or its proximate metabolites may serve as cotransmitters with norepinephrine in the adrenergic nervous system. (35 Refs.) Record Date Created: 19970328

18/7/84 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 08164191 94255188 PMID: 8196925

Control of eyelid retraction associated with Graves' disease with botulinum A toxin.

Biglan AW

Department of Ophthalmology, University of Pittsburgh School of Medicine, Pa.

Ophthalmic surgery (UNITED STATES) Mar 1994, 25 (3) p186-8, ISSN 0022-023X

Journal Code: OIC Languages: ENGLISH Document type: Journal Article Record type:

Completed

Two patients had satisfactory control of eyelid retraction associated with thyroid orbitopathy with repeated treatment of the levator palpebrae superioris muscle with botulinum A toxin. The effects of the toxin lasted for 3 to 4 months. Record Date Created: 19940630

18/7/88 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 08072229 93228655 PMID: 8471065

Tissue- and subunit-specific regulation of G-protein expression by hypo- and hyperthyroidism.

Michel-Reher MB; Gross G; Jasper JR; Bernstein D; Olbricht T; Brodde OE; Michel MC

Department of Medicine, University of Essen, Germany.

Biochemical pharmacology (ENGLAND) Apr 6 1993, 45 (7) p1417-23, ISSN 0006-2952

Journal Code: 924 Contract/Grant No.: HL 38741, HL, NHLBI Languages: ENGLISH

Document type: Journal Article Record type: Completed

Thyroid hormone status has profound effects on signal transduction in various tissues throughout the body. Therefore, we quantified the signal transducing G-proteins in the rat heart, cerebral cortex, vas deferens and liver by immunoblotting and pertussis toxin labeling in response to chemically induced hypothyroidism (treatment with propylthiouracil) and hyperthyroidism (treatment with triiodothyronine). Levels of the pertussis toxin (PTX) substrates Gi alpha and Go alpha in the heart and vas deferens were inversely correlated with thyroid hormone levels, i.e. Gi alpha and Go alpha were decreased or unchanged in hyperthyroid rats and increased in hypothyroid rats compared to control animals. The cerebral cortex and liver expression of PTX substrates Gi alpha and Go alpha was not affected by changes in thyroid hormone. Regulation of Gs alpha protein was more complex in that Gs alpha was unaffected in the other tissues tested. Expression of G-protein beta-subunits was not affected by thyroid status in the heart, liver, or cerebral cortex. Our results suggest that tissue- and G-protein-specific factors are involved in the regulation of G-protein subunits by thyroid hormone. Moreover, cardiac expression of Gs alpha is upregulated by increases or decreases in the normal level of thyroid hormone. Record Date Created: 19930510

18/7/93 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 07947261 94041171 PMID: 8225200

Regulation of calcitonin secretion in vitro.

Raue F; Zink A; Scherubl H

Abt. Innere Medizin I, Endokrinologie und Stoffwechsel, Universitat Heidelberg, Germany.

Hormone and metabolic research (GERMANY) Sep 1993, 25 (9) p473-6, ISSN 0018-5043

Journal Code: GBD Languages: ENGLISH Document type: Journal Article; Review; Review,

Tutorial Record type: Completed

The concentration of extracellular calcium rightly regulates calcitonin secretion by calcium influx through dihydropyridine-sensitive voltage-dependent calcium channels; the result is an increase in intracellular calcium. There also exists a cAMP-dependent pathway of calcitonin release activated by glucagon or growth hormone releasing hormone. In thyroid C-cells, as in all cells, there is dual regulation of adenylate cyclase, mediated by inhibitory or stimulatory G proteins; glucagon stimulated cAMP production can be inhibited by somatostatin via pertussis toxin sensitive inhibitory G proteins. Somatostatin inhibits not only cAMP dependent but also calcium-dependent calcitonin secretion. Furthermore, somatostatin inhibits voltage dependent calcium channel currents thereby lowering cytosolic calcium. These actions also involve a pertussis toxin - sensitive inhibitory G protein but they occur independently of changes in the cytosolic cAMP concentration. Thus multiple interactions between second messenger systems at different cellular levels modulate calcitonin secretion. (30 Refs.) Record Date Created: 19931217

18/7/94 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 07911503 93145899 PMID: 8381075

Overexpression of the intact thyrotropin receptor in a human thyroid carcinoma cell line.

Namba H; Yamashita S; Usa T; Kimura H; Yokoyama N; Izumi M; Nagataki S

Department of Cell Physiology, Nagasaki University School of Medicine, Japan.

Endocrinology (UNITED STATES) Feb 1993, 132 (2) p839-45, ISSN 0013-7227 Journal

Code: EGZ Languages: ENGLISH Document type: Journal Article Record type: Completed

Although thyrotropin is known to regulate thyroid cell differentiation and proliferation, human thyroid carcinoma cells are relatively insensitive or resistant to TSH stimulation. The expression levels of TSH receptor are significantly lower in carcinoma tissues than in normal tissues. Furthermore, in vitro human thyroid cell growth is not regulated by TSH itself. We, therefore, isolated neomycin-resistant stable human thyroid carcinoma cell (WRO cell) transfectants overexpressing intact human TSH receptor to evaluate the functional role of TSH receptor on carcinoma cells. Southern blot analysis confirmed incorporation and amplification of human TSH receptor complementary DNA sequences into genomic DNA. Northern gel analysis and reverse transcriptase-polymerase chain reaction analysis revealed the presence of specific TSH receptor messenger RNA (4.0 kilobases), and the specific binding and the affinity of [125I]TSH on stably transfected WRO cells were demonstrated compared to wild type. Nevertheless, impaired cAMP production to transfectants by TSH was observed. cAMP production was confirmed after stimulation of both wild type and transfectants by forskolin, cholera toxin, and isoproterenol. In contrast, TSH could affect the cytoplasmic calcium mobilization immediately after the addition of TSH to WRO transfectants. These results suggest that the impairment of TSH action on human thyroid carcinoma cells is not due to a major structural abnormality of the TSH receptor, reduction

In the receptor number, or receptor affinity, but much more likely due to SH receptor-guanyl nucleotide-binding protein coupling defect. Record Date Created: 1997/03/02

20/6/1 10358931 20000569 PMID: 10532769

Strabismus surgery among aged medicare beneficiaries. Dec 1997

20/6/2 10021711 99082623 PMID: 9865104

[Bilateral laryngeal movement disorder and synkinesia: value of botulism toxin. Apropos of a case] Trouble de la mobilité laryngée bilatérale et syncinésies: intérêt de la toxine botulique. A propos d'un cas. 1998

20/6/3 08164191 94255188 PMID: 8196925

Control of eyelid retraction associated with Graves' disease with botulinum A toxin. Mar 1994

20/6/4 07433158 91214931 PMID: 1902375

Management of dysthyroid eye disease. Apr 1991

20/6/5 07111148 94157131 PMID: 8113438

Botulinum toxin type A in upper lid retraction of Graves' ophthalmopathy. Dec 1993

20/6/6 06668510 91032399 PMID: 2226978

Thyroid eye disease. 1990

20/6/7 06668494 91032402 PMID: 2226981

Botulinum toxin therapy in dysthyroid strabismus. 1990

20/6/8 06275988 87108295 PMID: 3804629

Botulinum in the treatment of adult motility disorders. Winter 1986

20/6/9 06220002 86204801 PMID: 3703521

Botulinum toxin for the treatment of dysthyroid ocular myopathy. Apr 1986

20/6/10 06145851 86084827 PMID: 3841096

Botulinum chemodenervation for strabismus and other disorders. Winter 1985

20/6/11 05312485 89385497 PMID: 2779991

Botulinum toxin therapy of eye muscle disorders. Safety and effectiveness. American Academy of Ophthalmology. Sep 1989

20/6/12 05259551 90199325 PMID: 3273259

[The use of botulinum toxin in endocrine exophthalmos] Utilisation de la toxine botulinique dans les exophtalmies endocriniennes. 1988

20/6/13 05204825 89026356 PMID: 3179055

Botulinum toxin. Aug 1988

20/6/14 05131078 87070940 PMID: 3466462

Diplopia in thyroid eye disease. 1986

20/6/15 04905297 84225515 PMID: 6676980

Saccadic velocity measurements in strabismus. 1983

20/6/16 04576471 85026678 PMID: 6489104

Injection treatment of endocrine orbital myopathy. Aug 15 1984

20/7/6 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001
Dialog Corporation. All rts. reserv.
06668510 91032399 PMID: 2226978

Thyroid eye disease.

Elston JS

Eye (ENGLAND) 1990, 4 (Pt 4) pvii, ISSN 0950-222X

Journal Code: EYE

24/7/1 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
07433158 91214931 PMID: 1902375

Management of dysthyroid eye disease.

Fells P

Moorfields Eye Hospital, London.

British journal of ophthalmology (ENGLAND) Apr 1991, 75 (4) p245-6, ISSN 0007-1161

Journal Code: AZK Languages: ENGLISH Document type: Journal Article; Review; Review,
Tutorial Record type: Completed (10 Refs.) Record Date Created: 19910605

24/7/2 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
06668510 91032399 PMID: 2226978

Thyroid eye disease.

Elston JS

Eye (ENGLAND) 1990, 4 (Pt 4) pvii, ISSN 0950-222X Journal Code: EYE

Languages: ENGLISH Document type: Editorial Record type: Completed Record Date
Created: 19901205

24/7/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
06668494 91032402 PMID: 2226981

Botulinum toxin therapy in dysthyroid strabismus.

Lyons CJ; Vickers SF; Lee JP

Moorfields Eye Hospital, London.

Eye (ENGLAND) 1990, 4 (Pt 4) p538-42, ISSN 0950-222X Journal Code: EYE

Languages: ENGLISH Document type: Journal Article Record type: Completed

We report our experience with the use of Botulinum toxin injection in 38 patients (64 injections)
with severe dysthyroid strabismus. Three quarters of the injections led to a decrease in the angle
of the squint by a mean 75% of the initial deviation. The average duration of effect was two
months. Twenty six patients went on to surgery after stabilisation of their squint and endocrine
status. Six patients achieved a stable long-term result with Botulinum toxin only. We suggest
these results of treatment of early dysthyroid myopathy are more consistent with the
characteristics of inflammatory spasm than contracture. The value of Botulinum toxin as a
temporary means of maintaining binocularity in these young patients is discussed. Record Date
Created: 19901205

24/7/4 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
06275988 87108295 PMID: 3804629

Botulinum in the treatment of adult motility disorders.

Hoffman RO; Helveston EM

International ophthalmology clinics (UNITED STATES) Winter 1986, 26 (4) p241-50, ISSN
0020-8167 Journal Code: GTZ Languages: ENGLISH Document type: Journal Article

Record type: Completed Record Date Created: 19870326

24/7/5 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
06220002 86204801 PMID: 3703521

Botulinum toxin for the treatment of dysthyroid ocular myopathy.

Dunn WJ; Arnold AC; O'Connor PS

Ophthalmology (UNITED STATES) Apr 1986, 93 (4) p470-5, ISSN 0161-6420 Journal
Code: O15 Languages: ENGLISH Document type: Journal Article Record type: Completed

Eight consecutive patients with acquired deviations due to dysthyroid ocular myopathy were
injected with botulinum A toxin for relief of their diplopia. Seven patients were acute in the onset of
symptoms and one was chronic. All showed improvement in motility and experienced a reduction
if not total relief of their symptoms. Six patients required reinjection. Complications were limited to
transient ptosis, transient involvement of adjacent muscles and transient but prolonged paralysis
that eventually resolved. No systemic complications were noted. We conclude that
chemodenervation with botulinum A toxin may have a role in the management of dysthyroid
ocular myopathy not amenable to prism treatment and may act as an adjunct to or eliminate the
need for surgical correction in some patients. Record Date Created: 19860613

24/7/6 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
05204825 89026356 PMID: 3179055

Botulinum toxin.

Kowal L

Australian and New Zealand journal of ophthalmology (AUSTRALIA) Aug 1988, 16 (3)
p264-6, ISSN 0814-9763 Journal Code: ANZ Languages: ENGLISH Document type: Journal
Article Record type: Completed Record Date Created: 19881220

24/7/7 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
05131078 87070940 PMID: 3466462

Diplopia in thyroid eye disease.

Fells P; McCarty B

Transactions of the ophthalmological societies of the United Kingdom (ENGLAND) 1986,
105 (Pt 4) p413-23, ISSN 0078-5334 Journal Code: WA1 Languages: ENGLISH Document
type: Journal Article Record type: Completed
Record Date Created: 19870122

Languages: ENGLISH Document type: Editorial Record
type: Completed Record Date Created: 19901205

20/7/13 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001

Dialog Corporation. All rts. reserv.

05204825 89026356 PMID: 3179055

Botulinum toxin.

Kowal L

Australian and New Zealand journal of ophthalmology
(AUSTRALIA) Aug 1988, 16 (3) p264-6, ISSN 0814-9763
Journal Code: ANZ Languages: ENGLISH Document type:
Journal Article Record type: Completed Record Date Created:
19881220

24/7/8 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
04576471 85026678 PMID: 6489104

Injection treatment of endocrine orbital myopathy.

Scott AB

Documenta ophthalmologica (NETHERLANDS) Aug 15 1984, 58 (1) p141-5, ISSN 0012-
4486 Journal Code: EBF

Contract/Grant No.: EY02106, EY, NEI Languages: ENGLISH Document type: Journal Article
Record type: Completed

Eight patients with endocrine orbital myopathy received botulinum toxin injection of extraocular
muscles for strabismus or injections of the levator for lid retraction. Strabismus of 25 prism iopters
or less, especially during early stages of eye muscle involvement, responded well to injection with
realignment and, probably, with avoidance of fixed muscle shortening. Long-standing strabismus,
large angles, and lid retraction responded less well. Record Date Created: 19841123

Set Items Description

Ref	Items	RT	Index-term
E1	1		TETANUCLEATE
E2	1		TETANUKSEN
E3	13902	3	*TETANUS
E4	71		TETANUS --BLOOD --BL
E5	5		TETANUS --CEREBROSPINAL FLUID --CF
E6	30		TETANUS --CHEMICALLY INDUCED --CI
E7	12		TETANUS --CLASSIFICATION --CL
E8	393		TETANUS --COMPLICATIONS --CO
E9	21		TETANUS --CONGENITAL --CN
E10	338		TETANUS --DIAGNOSIS --DI
E11	3		TETANUS --DIET THERAPY --DH
E12	523		TETANUS --DRUG THERAPY --DT

Ref	Items	Type	RT	Index-term
R1	13902	3		*TETANUS
R2	4515	X		DC=C1.252.410.90.217.864. (TETANUS)
R3	904	R	6	TRISMUS
R4	2533	B	7	CLOSTRIDIUM INFECTIONS

Ref	Items	Type	RT	Index-term
R1	2533	7		*CLOSTRIDIUM INFECTIONS
R2	2533	X		DC=C1.252.410.90.217. (CLOSTRIDIUM INFECTIONS)
R3	122	B	9	BACILLACEAE INFECTIONS
R4	1785	N	10	BOTULISM
R5	3997	N	10	ENTEROCOLITIS, PSEUDOMEMBRANOUS
R6	279	N	3	ENTEROTOXEMIA
R7	1368	N	2	GAS GANGRENE
R8	13902	N	3	TETANUS

S1 13902 "TETANUS"
S2 2533 "CLOSTRIDIUM INFECTIONS"
S3 90754 THYROID
S4 38 S1 AND S3
S5 2 S2 AND S3

Ref	Items	RT	Index-term
E1	753		THYROTROPIC
E2	2		THYROTROPICAL
E3	25841	8	*THYROTROPIN
E4	167		THYROTROPIN --ADMINISTRATION AND DOSAGE --AD
E5	30		THYROTROPIN --ADVERSE EFFECTS --AE
E6	17		THYROTROPIN --ANALOGS AND DERIVATIVES --AA
E7	896		THYROTROPIN --ANALYSIS --AN
E8	255		THYROTROPIN --ANTAGONISTS AND INHIBITORS --AI
E9	288		THYROTROPIN --BIOSYNTHESIS --BI
E10	10117		THYROTROPIN --BLOOD --BL
E11	12		THYROTROPIN --CEREBROSPINAL FLUID --CF
E12	6		THYROTROPIN --CHEMICAL SYNTHESIS --CS

S6 25841 "THYROTROPIN"
S7 19 S6 AND (S1 OR S2)
S8 30150 "THYROID GLAND"

Ref	Items	RT	Index-term
E1	96		THYROID FUNCTION TESTS --VETERINARY --VE
E2	0	1	THYROID GALACTOSYLTRANSFERASE
E3	30150	5	*THYROID GLAND

E4	595		THYROID GLAND --ABNORMALITIES --AB
E5	1135		THYROID GLAND --ANALYSIS --AN
E6	992		THYROID GLAND --ANATOMY AND HISTOLOGY --AH
E7	672		THYROID GLAND --BLOOD SUPPLY --BS
E8	369		THYROID GLAND --CHEMISTRY --CH
E9	2573		THYROID GLAND --CYTOLOGY --CY
E10	1		THYROID GLAND --DIAGNOSIS --DI
E11	5066		THYROID GLAND --DRUG EFFECTS --DE
E12	601		THYROID GLAND --EMBRYOLOGY --EM

Ref	Items	Type	RT	Index-term
R1	30150	5		*THYROID GLAND
R2	30150	X		DC=A6.407.900. (THYROID GLAND)
R3	221	R	3	STRUMA OVARII
R4	11014	R	3	THYROIDECTOMY
R5	217	R	3	ULTIMOBRANCHIAL BODY
R6	5409	B	30	ENDOCRINE GLANDS

S9 17616 CLOSTRID?
S10 7 S8 AND S9
S11 133 PHOSPHOLIPASE AND NEUROTOXIN
S12 27833 THYROX?
S13 2 S9 AND S12
S14 6426 BOTULI?
S15 0 S14 AND S12
S16 0 S14 AND S8
S17 18 S3 AND S14

4/6/1 11245935 21195143 PMID: 11298123
Intradermal skin test with diabetes specific antigens in patients with type 1 diabetes. Mar 2001

4/6/2 10726207 20373526 PMID: 10916814
[Preventive activities in primary health care: identifying the agreement among evidence-based guidelines] Actividades preventivas en atencion primaria: identificacion de areas de concordancia entre guias de practica clinica basadas en la evidencia. 2000

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Muscle reinnervation in hypothyroid rats. 1996

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Detection of novel carbohydrate binding activity of interleukin-1. Feb 12 1999

4/6/5 09732112 98204670 PMID: 9545109
Induction of oral tolerance in human autoimmune thyroid disease. Mar 1998

4/6/6 09716948 98187354 PMID: 9526606
Preliminary studies with recombinant chorionic gonadotropin beta-subunit produced in Escherichia coli for use as an antigen in a birth control vaccine. Mar 1998

4/6/7 09511341 95156316 PMID: 7853237
Effects of thyroid hormone on fast- and slow-twitch skeletal muscles in young and old rats. Nov 15 1994

4/6/8 09419224 97479154 PMID: 9337806
Midlife periodic health exam in the primary care practice. Oct 1997

4/6/9 09175752 96429836 PMID: 8832986
Elevated serum prolactin or elevated prolactin/cortisol ratio are associated with autoimmune processes in systemic lupus erythematosus and other connective tissue diseases. Mar 1996

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The pathogenicity of spontaneously-occurring thyroglobulin-reactive T lymphocytes from BB/WOR rats. 1996

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Activation of T lymphocyte subsets by synthetic TSH receptor peptides and recombinant glutamate decarboxylase in autoimmune thyroid disease and insulin-dependent diabetes. Apr 1995

4/6/12 08365713 95213731 PMID: 7699384
Response of fast muscle innervation to hypothyroidism. Dec 1 1994

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[Evaluation on our procedure for autotransplantation of parathyroid glands by the intact-PTH] Aug 1993

4/6/14 07651909 93027039 PMID: 1408659
The time course of thyroid-hormone-induced changes in the isotonic and isometric properties of rat soleus muscle. Jul 1992

4/6/15 07355645 90368987 PMID: 2168443
Thyroid-stimulating antibody activity between different immunoglobulin G subclasses. Sep 1990

4/6/16 07344123 90192059 PMID: 2138281
The rate of tetanic relaxation is correlated with the density of calcium ATPase in the terminal cisternae of thyrotoxic skeletal muscle. Jan 1990

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Screening practices of family physicians: a comparison of STFM and AAFP members. Sep-Oct 1987

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Characteristics and bioefficacy of monoclonal antigonadotropin releasing hormone antibody. Mar 1985

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Probing the normal and autoimmune B cell repertoire with Epstein-Barr virus. Frequency of B cells producing monoreactive high affinity autoantibodies in patients with Hashimoto's disease and systemic lupus erythematosus. Dec 15 1988

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Enhancement of antigonadotropin response to the beta-subunit of ovine luteinizing hormone by carrier conjugation and combination with the beta-subunit of human chorionic gonadotropin. Jul 1986

4/6/22 05330203 90033436 PMID: 2806615
Antibody response and characteristics of antibodies in women immunized with three contraceptive vaccines inducing antibodies against human chorionic gonadotropin. Nov 1989

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Affinity purification of IgG subclasses and the distribution of thyroid auto-antibody reactivity in Hashimoto's thyroiditis. Jul 1989

4/6/24 05235053 88154038 PMID: 3257970
Microsomal antigen-reactive lymphocyte lines and clones derived from thyroid tissue of patients with Graves' disease. Apr 1988

4/6/25 04893804 80227723 PMID: 7391005
Interaction of fragments B and C of tetanus toxin with neural and thyroid membranes and with gangliosides. Jul 10 1980

4/6/26 04805868 82247718 PMID: 6179073
[Possible relation between the immune response and thymus-dependence of the immunizing antigens in children in an endemic goiter region] O vozmozhnoi svyazi mezhdu immunnym ovetom i timuszavisimost'iu immuniziruushchikh antigenov u detei v ochage zobnoi endemii. May-Jun 1982

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Gangliosides, the thyrotropin receptor, and autoimmune thyroid disease. 1984

4/6/28 04713242 80150428 PMID: 6244725
Thyrotropin receptors and gangliosides. 1980

4/6/29 04661467 84263155 PMID: 6378767
Defective regulation of the immune response to tetanus toxoid in Hashimoto's disease. Jul 1984

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Characterization of monoclonal antibody 3G5 and utilization of this antibody to immobilize pancreatic islet cell gangliosides in a solid phase radioassay. Apr 1984

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Thyroid function in tetanus. Jul 1981

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Structure/function studies of receptors for thyrotropin and tetanus toxin: lipid modulation of effector binding to the glycoprotein receptor component. Jul 14 1978

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Tetanus toxin and thyrotropin interactions with rat brain membrane preparations. May 25 1979

4/6/34 03389313 79127176 PMID: 217603

Tetanus toxin interactions with the thyroid: decreased toxin binding to membranes from a thyroid tumor with a thyrotropin receptor defect and in vivo stimulation of thyroid function. Mar 1978

Use sub-linguale aux molecules organiques. Les limites du role de la sub-linguale dans la vaccination par aerosols 1975

Tetanus toxin interactions with thyroid plasma membranes. Implications for structure and function of tetanus toxin receptors and potential pathophysiological significance. Jun 25 1977

4/6/35 03345587 76185875 PMID: 131619

[Permeability of sublingual mucosa to organic molecules. Limited role of sublingual absorption in aerosol vaccinations] La permeabilite de la

4/6/36 03008262 76102749 PMID: 813223

Isomunization against human chorionic gonadotropin with conjugates of processed beta-subunit of the hormone and tetanus toxoid. Jan 1976

4/6/38 01235275 70276257 PMID: 4916539

Immunosuppressive therapy for the eye changes of Graves' disease. Sep 1970

4/6/37 02679440 77187913 PMID: 193853

4/7/2 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 10726207 20373526 PMID: 10916814

[Preventive activities in primary health care: identifying the agreement among evidence-based guidelines] Actividades preventivas en atencion primaria: identificacion de areas de concordancia entre guias de practica clinica basadas en la evidencia.

Gosalbes Soler V; Marquez Calderon S; Maiques Galan A; Latour Perez J; Bernal Delgado E; Puig Barbera J; Arranz Lazaro M

Unidad Docente de Medicina Familiar y Comunitaria, Valencia. gosalbesvic@gua.es
Medicina clinica (SPAIN) 2000, 114 Suppl 2 p88-92, ISSN 0025-7753 Journal Code: LTQ
Languages: SPANISH Document type: Journal Article Record type: Completed

BACKGROUND: The purpose of this article is to identify the agreement among evidence-based guidelines about recommendations on preventive activities in low risk adults. METHODS: For which we identified, from the 1996 US Task Force on Preventive Services Guide those preventives activities classified like A or B (recommendation in favour of provision) and like D or E (recommendation against provision), excepting those D and E recommendations based on descriptive studies or expert opinions. Both preventive activities aimed at pregnant women and children and those which are not applicable to our context were excluded. Selected preventive services were compared with the range of age, sex and periodicity in which agreement with the recommendations of American College of Physicians and Canadian Task Force on Preventive Services existed. RESULTS: We found the following agreements. Screening activities. In favour: screening for hypercholesterolemia, hypertension, breast cancer, colorectal cancer, uterine cervix cancer, rubella, visual and hearing impairment and problem drinking. Against: cancer of prostate, lung, bladder and thyroid, and asymptomatic bacteriuria. Counseling activities. In favour: smoking, motor-vehicles injuries, alcohol consumption, unintended pregnancy. Immunizations and quinioprophylaxis. In favour: Vaccines for influenza, tetanus-diphtheria, hepatitis B and measles-mumps-rubella. Postexposure prophylaxis to hepatitis A, hepatitis B, meningococcal, rabies and tetanus. CONCLUSIONS: We see then, that a high degree in agreement among the main guidelines exists; about the preventive activities to perform in Primary Health Services, nevertheless we observed low fulfillment of certain preventive activities in Primary Health Services, different barriers for the accomplishment from these activities were described. Record Date Created: 20000919

4/7/5 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09732112 98204670 PMID: 9545109

Induction of oral tolerance in human autoimmune thyroid disease.

Lee S; Scherberg N; DeGroot LJ

Department of Medicine, The University of Chicago, Illinois, USA.

Thyroid (UNITED STATES) Mar 1998, 8 (3) p229-34, ISSN 1050-7256 Journal Code: BJW
Languages: ENGLISH Document type: Clinical Trial; Journal Article; Randomized Controlled Trial Record type: Completed

Our laboratory has reported suppression of experimental autoimmune thyroiditis in mice by oral feeding with antigen. Based on these data, we considered it possible that oral feeding of animal thyroglobulin (TG) might induce tolerance to antigen in human autoimmune thyroid disease (AITD). Thirteen patients receiving thyroid hormone replacement with synthetic thyroxine (T4) (five patients with Graves' disease, treated with radiiodine 4 to 11 years ago and eight patients with Hashimoto's thyroiditis) were randomly assigned to a test group (switched to replacement with desiccated thyroid from porcine thyroids) and a control group (maintained on synthetic T4). Humoral and cellular immunologic parameters were evaluated in addition to clinical parameters before and every 3 months after the onset of study for a year. At the onset of study, there was no difference in clinical parameters, or humoral and cellular immunity to thyroid autoantigens, except a finding that one thyroid peroxidase (TPO) peptide (100 approximately 119) appeared to stimulate peripheral blood mononuclear cells (PBMC) during in vitro microproliferation assay more in the test group than control group ($p = 0.051$ by t test). Additionally, almost all of TPO and thyrotropin receptor extracellular domain (TSHR) peptides were slightly more stimulatory to PBMC from the test group than the control group, although this was not statistically significant. After treatment, all variables were analyzed at each time point between groups (t test), and also were analyzed over time in each group (analysis of variance, ANOVA). Among the clinical parameters, thyrotropin (TSH) levels were unchanged and equal. Total serum T4 levels ($p < 0.05$ at 6 and 12 months after treatment) and free thyroxine indices (FT4I) ($p < 0.05$ at all time points after treatment) were lower in the test group than the control group. This is an expected result of treatment with desiccated thyroid. We found no change over time nor any difference between groups at time points for titers of antibodies to thyroid autoantigens, ie, human TG, human TPO, and recombinant human TSHR from *Escherichia coli*. However, cellular immunity, measured by in vitro microproliferation of PBMC to peptides of TPO or TSHR, showed significant differences between groups. At 12 months, stimulatory indices (SI) of PBMC to six peptides, containing the indicated amino acids (764 approximately 95, 100 approximately 119, 110 approximately 129, 261 approximately 275, 441 approximately 448, 708 approximately 727) of 10 TPO peptides, and one peptide (145 approximately 163) of 14 TSHR peptides were lower in the test group than control group ($p < 0.05$). SI of PBMC to phytohemagglutinin, purified protein derivative from mycobacteria, and tetanus toxoid were not different between groups nor changed over time in any group. In conclusion, treating patients with AITD with an antigen related to the autoantigen TG did not produce changes in humoral immunity parameters, while cellular immunity to certain peptides were apparently suppressed. While the results are both surprising and intriguing, we need more evidence to justify the use of autoantigen as a form of immunospecific therapy in patients with AITD. Record Date Created: 19980515

4/7/7 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09511341 95156316 PMID: 7853237

Effects of thyroid hormone on fast- and slow-twitch skeletal muscles in young and old rats.

Larsson L; Li X; Teresi A; Salvati G

Department of Clinical Neurophysiology, Karolinska Hospital, Stockholm, Sweden.

Journal of physiology (ENGLAND) Nov 15 1994, 481 (Pt 1) p149-61, ISSN 0022-3751

Journal Code: JQV Languages: ENGLISH Document type: Journal Article Record type: Completed

1. The effects of 4 weeks of thyroid hormone treatment on contractile, enzyme-histochemical and morphometric properties and on the myosin isoform composition were compared in the slow-twitch soleus and the fast-twitch extensor digitorum longus (EDL) muscle in young (3-6 months) and old (20-24 months) male rats. 2. In the soleus of untreated controls, contraction and half-relaxation times of the isometric twitch increased by 19-32% with age. The change in contractile properties was paralleled by an age-related increase in the proportions of type I fibres and type I myosin heavy chain (MHC) and slow myosin light chain (MLC) isoforms. 3. In the EDL of controls, contraction and half-relaxation times were significantly prolonged (21-38%) in the post-tetanus twitch in the old animals. No significant age-related changes were observed in enzyme-histochemical fibre-type proportions, although the number of fibres expressing both type IIA and IIB MHCs and of fibres expressing slow MLC isoforms was increased in the old animals. 4. Serum 3,5,3',5'-tetraiodothyronine (T4) levels were lower (34%) in the old animals; but the primary byproduct of T4, 3,5,3'-triiodothyronine (T3), did not differ between young and old animals. 5. The effects of 4 weeks of thyroid hormone treatment were highly muscle specific, and were more pronounced in soleus than in EDL, irrespective of animal age. In the soleus, this treatment shortened the contraction and half-relaxation times by 35-57% and decreased the number of type I fibres by 66-77% in both young and old animals. In EDL, thyroid hormone treatment significantly shortened the contraction time by 24%, but the change was restricted to the old animals. 6. In conclusion, the ability of skeletal muscle to respond to thyroid hormone treatment was not impaired in old age and the age-related changes in speed of contraction and enzyme-histochemical properties and myosin isoform compositions were diminished after thyroid hormone treatment in both the soleus and EDL. Record Date Created: 19950314

4/7/12 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 08365713 95213731 PMID: 7699384

Response of fast muscle innervation to hypothyroidism.

Cuppini R; Sartini S; Ambrogini P; Gallo G

Istituto di Anatomia e Fisiologia, Universita di Urbino, Italy.

Journal of the neurological sciences (NETHERLANDS) Dec 1 1994, 127 (1) p107-13, ISSN 0022-510X Journal Code: JBJ Languages: ENGLISH Document type: Journal Article Record type: Completed

The early period of motor innervation development is characterized by multiple innervation of muscle cells. This transitory state in rat extensor digitorum longus (edl) muscle is normally concluded at weaning when a 1:1 ratio between nerve endings and muscle cells is reached. Motor innervation of edl muscle in rats made hypothyroid after weaning was studied in three ways: electrophysiology (intracellular recordings of muscle postsynaptic potentials) was carried out to study neuromuscular transmission; silver impregnation of terminal axons to observe sprouting; force production in twitch and tetanus following direct muscle stimulation and nerve stimulation. A number of multiply innervated muscle cells was found in hypothyroid rats following two months of treatment. This finding seems to be related to the appearance of nodal sprouting in motor axons. No sign of denervated end-plates was found. Twitch and tetanus tension were smaller than in controls, but they were bigger when referred to unitary muscle mass. Time course of twitch, particularly half relaxation, was slowed in muscles of hypothyroid rats. These findings suggest that plastic processes occur in muscle innervation of rats made hypothyroid after weaning. Therefore, thyroid hormones play a role in stabilizing motor innervation not only during development, but also in adults. Record Date Created: 19950428

4/7/13 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 07938907 93390517 PMID: 8377760

[Evaluation on our procedure for autotransplantation of parathyroid glands by the intact-PTH]

Ueda M; Funahashi H; Sato Y; Kato M; Takagi H

Second Department of Surgery, Nagoya University, School of Medicine, Japan.

Nippon Geka Gakkai zasshi (JAPAN) Aug 1993, 94 (8) p840-6, ISSN 0301-4894 Journal

Code: NGG Languages: JAPANESE Document type: Journal Article Record type: Completed
In thyroid cancer surgery since 1978 we have made parathyroidal autotransplantation which has been to resect the entire parathyroid, cut fine to mud and autotransplant into greater pectoral muscle. To evaluate our procedure and consider the mechanism of hypocalcemia and tetanus following surgery, we examined the intact-PTH recovered to about 80% of its preoperative value on 14-postoperative-days and thereafter remained almost constant. It remained below the sensitivity of measurement from immediately to 3-postoperative-days, and with our procedure, parathyroid was considered to be totally resected. Comparison between 2 (6 cases) and more autotransplanted glands (11 cases) revealed that the former showed slightly later functional recovery, but recovered to the almost same extent as the other 21-postoperative-days. Comparison between the group requiring calcium supplement therapy (12 cases) and the otherwise (5 cases) revealed almost the same course of recovery. Thus, our procedure seems to enable us to make satisfactory functional preservation and expect functional recovery by 2-gland

autotransplantation at least. The supplement therapy was considered to be detrimental to the take of autotransplanted glands and functional recovery, and no correlation was noted between the onset of tetanus and intact-PTH. Record Date Created: 19931020

4/7/17 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 06343059 88056008 PMID: 3678672

Screening practices of family physicians: a comparison of STFM and AAFP members.

Resnicow K; Schorow M; Bloom HG; Massad R; Coll-Barth M

American Health Foundation, New York City.

Family medicine (UNITED STATES) Sep-Oct 1987, 19 (5) p341-5, ISSN 0742-3225

Journal Code: FAL Languages: ENGLISH Document type: Journal Article Record type: Completed

The screening practices of 146 members of the Society of Teachers of Family Medicine (STFM) and 129 members of the American Academy of Family Physicians (AAFP) are compared. The screening practices of physicians from the two organizations were generally similar for psychosocial and behavioral problems, many forms of cancer, and numerous other conditions considered for inclusion in the routine periodic screening of asymptomatic individuals. However, for numerous diseases and tests, the screening practices of physicians from the two groups were significantly different. AAFP physicians were more likely to screen for lung and skin cancer, thyroid dysfunction, diabetes, and anemia, AAFP physicians were more likely to utilize chest x-ray, ECG, urinalysis, and SMA 6/12. STFM physicians were more likely to perform gonococcal culture and tetanus-diphtheria immunization as well as to inquire about seat belt use. Three variables were found to predict physician screening practices as well as to account for the differences found between physicians drawn from the two organizations: completion of a residency in family medicine, year of graduation from medical school, and number of patients seen per week. Physicians reported practices were compared with recommendations in the major critical reviews: Frame and Carlson, Breslow and Sommers, the Canadian Task Force, and the American Cancer Society. For a number of tests and diseases physicians' reported practices were divergent with recent recommendations. Record Date Created: 19871221

4/7/20 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 05639197 88043483 PMID: 3118511

Temporal analysis of dithiobiuret neurotoxicity in rats and assessment of potential nonneural causes.

Williams KD; Miller MS; Boysen BG; Peterson RE

School of Pharmacy, University of Wisconsin, Madison 53706.

Toxicology and applied pharmacology (UNITED STATES) Nov 1987, 91 (2) p212-21, ISSN 0041-008X Journal Code: VWO Contract/Grant No.: 1-K04-ES00098, ES, NIEHS; ES01906, ES, NIEHS Languages: ENGLISH Document type: Journal Article Record type: Completed

To evaluate the hypothesis that depressed neuromuscular transmission causes dithiobiuret (DTB)-induced muscle weakness in rats, the temporal development of impaired treadmill performance and deficits in the nerve-elicited muscle contractions were compared during daily treatment with the toxicant (DTB, 1 mg/kg/day X 6 days). Diminished treadmill test performance after 4 days of treatment marked the initial detection of impaired motor function. At this time fading (loss of tension during tetanus) of gastrocnemius contractions elicited in response to 100-Hz sciatic nerve stimulation occurred in DTB-treated rats but not in controls. After 5 and 6 days of treatment, treadmill failure became complete, tetanic fade worsened dramatically, and peak contractile tension measured during trains of nerve stimulation (10-250 Hz) decreased progressively. Appearing by Day 6 were marked body weight loss, dehydration, hypothermia, and a depression in serum concentrations of thyroid hormones. Total oxygen content of the blood was not reduced at any time during treatment, and serum concentrations of glucose, sodium, potassium, calcium, chloride, and phosphorus in DTB-treated rats on Day 6 were similar to those of control animals. Therefore, hypoxia, hypoglycemia, or a serum electrolyte imbalance do not initiate or modulate the neuromuscular toxicity. Light microscopic evaluation of liver, kidney, lung, thyroid, and other organs in intoxicated rats was unremarkable and in skeletal muscles and selected sites of brain, spinal cord, and sciatic nerve no morphologically significant lesions were observed. Even when DTB-intoxicated rats were maintained in a state of flaccid muscle weakness for 5 continuous days, peripheral nerve lesions proximal to the intramuscular nerves were not detected. Thus, depressed neuromuscular transmission appears to be the primary cause of the flaccid muscle weakness and no evidence was obtained that nonneural effects of DTB initiate or modulate this effect. Record Date Created: 19871211

4/7/24 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 05235053 88154038 PMID: 3257970

Microsomal antigen-reactive lymphocyte lines and clones derived from thyroid tissue of patients with Graves' disease.

Fisfalen ME; DeGroot LJ; Quintans J; Franklin WA; Soltani K

Department of Medicine, University of Chicago, Illinois 60637.

Journal of clinical endocrinology and metabolism (UNITED STATES) Apr 1988, 66 (4) p776-84, ISSN 0021-972X Journal Code: HRB Contract/Grant No.: AM-13377, AM, NIADDK; CA19589, CA, NCI; DK-27384, DK, NIDDK

Languages: ENGLISH Document type: Journal Article Record type: Completed

Thyroid mononuclear cells (TMC) were maintained in long term cocultures with thyroid fibroblasts and thyroid epithelial cells from patients with Graves' disease, using medium supplemented with thyroid microsomal antigen (McAg) and IL-2. The TMC consisted predominantly of T4+ (CD4+, helper) and, to a lesser extent, T8+ (CD8+, cytotoxic/suppressor) lymphocytes, with a small number of macrophages and natural killer cells. The average T4+ to T8+ ratio was 3.2. From these cultures we obtained thyroid T cell lines and clones reactive to thyroid antigens. T Cell lines were tested in a microproliferation assay using thyroglobulin (Tg), McAg, tetanus toxoid, and IL-2. Of 14 lines from 6 patients, 2 proliferated in response to McAg when TMC plus thyroid fibroblasts were used as antigen-presenting cells. Clones of thyroid lymphocytes were obtained by culturing cells at limiting dilution with IL-2, McAg, and different types of autologous accessory cells.

Peripheral blood mononuclear cells and skin fibroblasts provided the best source of accessory cells, allowing near 100% cloning efficiency. Of 26 clones tested, 6 recognized McAg, 2 were Tg reactive, and 3 were autoreactive. All phenotyped clones were of the T4+ phenotype. Our method results in production of thyroid T cell lines and clones. The fibroblasts probably provided growth factors and/or collaborated with peripheral blood mononuclear cells as antigen-presenting cells. These lines and clones from patients with Graves' disease were predominantly helper T cells, in contrast to the previously demonstrated cytotoxic/suppressor cell predominance in cells from patients with Hashimoto's thyroiditis. This difference in cell function may help explain the differing clinical courses of these two closely related autoimmune thyroid diseases. The availability of long term microsomal antigen-specific T cell clones should allow careful analysis of the role these cells play in thyroid autoimmunity. Record Date Created: 19880419

4/7/25 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04893804 80227723 PMID: 7391005

Interaction of fragments B and C of tetanus toxin with neural and thyroid membranes and with gangliosides.

Morris NP; Consiglio E; Kohn LD; Habig WH; Hardegree MC; Helting TB

Journal of biological chemistry (UNITED STATES) Jul 10 1980, 255 (13) p6071-6, ISSN 0021-9258 Journal Code: HIV

Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19800928

4/7/27 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04760092 84253338 PMID: 6331133

Gangliosides, the thyrotropin receptor, and autoimmune thyroid disease.

Lacetti P; Tombaccini D; Aloj S; Grollman EF; Kohn LD

Advances in experimental medicine and biology (UNITED STATES) 1984, 174 p355-67, ISSN 0065-2598 Journal Code: 2LU Languages: ENGLISH Document type: Journal Article Record type: Completed

The thyrotropin (TSH) receptor has been proposed to be composed of a membrane glycoprotein and a membrane ganglioside, the former important in high affinity recognition, the latter vital for message coupling to the adenylate cyclase system. The present study used two approaches, formation of antireceptor monoclonal antibodies and reconstitution, to validate the model and further examine the role of the ganglioside. Three kinds of monoclonal antireceptor antibodies are defined. One group which inhibits TSH binding and TSH functions, i.e., TSH-stimulated adenylate cyclase activity, iodide uptake, and thyroid hormone release, is shown to be directed against the glycoprotein component of the receptor. The second group includes antibodies which mimic TSH in all stimulatory actions, are competitive agonists of TSH, are equivalent to thyroid stimulating antibodies in the sera of patients with Graves' disease, and are directed against the ganglioside component of the receptor. These stimulating monoclonal antibodies are directed against a minor ganglioside membrane component which fractionates as a disialoganglioside. When this ganglioside is incorporated into 1-8 thyroid cells which have a correlated ganglioside deficiency and TSH receptor defect, reconstitution of TSH stimulated adenylate cyclase activity occurs. Whereas the first group of antibodies inhibits TSH-stimulated function, they do not inhibit the stimulatory antibodies which mimic TSH, an observation consistent with the 2 component hypothesis of the receptor model. The third group of antibodies have a mix of properties from the first two groups and suggests that the TSH receptor in situ is an actual complex of the two components or that there are common carbohydrate determinants in the functional sites of each receptor component. Implications of a TSH receptor structure in which its ganglioside and glycoprotein components are in equilibrium with pools of free components and, in turn, components important for cholera toxin, tetanus toxin and interferon receptors are discussed. In regard to the pathogenesis of Graves' disease, the data indicate that thyroid stimulating autoantibodies are autoimmune equivalents of cholera toxin with respect to the importance of ganglioside function. Since antidiotype studies of antibodies against TSH confirm a structural relationship between receptors for thyrotropin, cholera toxin, and thyroid stimulating autoantibodies, the data establish an unequivocal role for the ganglioside in TSH receptor structure which facilitates interpretation of in vitro experiments aimed at understanding the mechanism of ganglioside-ligand interactions. Record Date Created: 19840823

4/7/28 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04713242 80150428 PMID: 6244725

Thyrotropin receptors and gangliosides.

Kohn LD; Consiglio E; DeWolf MJ; Grollman EF; Ledley FD; Lee G; Morris NP

Advances in experimental medicine and biology (UNITED STATES) 1980, 125 p487-503, ISSN 0065-2598 Journal Code: 2LU Languages: ENGLISH Document type: Journal Article; Review Record type: Completed (44 Refs.) Record Date Created: 19800530

4/7/29 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04661467 84263155 PMID: 6378767

Defective regulation of the immune response to tetanus toxoid in Hashimoto's disease.

Fawcett J; Hutton C; McLachlan SM; Clark F; Rees Smith B

Immunology (ENGLAND) Jul 1984, 52 (3) p525-8, ISSN 0019-2805 Journal Code: GH7

Languages: ENGLISH Document type: Journal Article Record type: Completed

The humoral immune response to tetanus toxoid has been studied in patients with Hashimoto's disease. Although the magnitude of the response was similar to that observed in normal subjects, the Hashimoto patients demonstrated an inability to regulate their levels of tetanus toxoid antibody. This apparent defect in the control of antibody synthesis may be an important factor in both the initiation and perpetuation of autoimmune thyroid disease. Record Date Created: 19840829

4/7/31 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 03639753 82119926 PMID: 7328074

Thyroid function in tetanus.
 Dastur FD; Shah J; Awatramani V; Shah JM; Pardiwalla BS; Nair KG; Mehta MN; Desai KB
 Journal of the Association of Physicians of India (INDIA) Jul 1981, 29 (7) p519-25, ISSN
 0004-5772 Journal Code: HG7 Languages: ENGLISH Document type: Journal Article
 Record type: Completed Record Date Created: 19820412

4/5/34 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
 03389313 79127176 PMID: 217603

Tetanus toxin interactions with the thyroid: decreased toxin binding to membranes from a thyroid tumor with a thyrotropin receptor defect and in vivo stimulation of thyroid function.
 Habig WH; Grollman EF; Ledley FD; Meldolesi MF; Aloj SM; Hardegree MC; Kohn LD
 Endocrinology (UNITED STATES) Mar 1978, 102 (3) p844-51, ISSN 0013-7227 Journal
 Code: EGZ Languages: ENGLISH Document type: Journal Article Record type: Completed
 Normal rat thyroid membranes adsorb neurotoxicity when incubated with purified tetanus toxin.
 Membranes from a rat thyroid tumor with a thyrotropin receptor defect adsorb very little
 neurotoxicity when similarly evaluated. This inability of the tumor membranes to adsorb
 neurotoxicity is correlated with a defect in their ability to bind both 125I-labeled tetanus toxin and
 [125I]iodothyrotropin. The effect of tetanus toxin on the release of radioiodine from the thyroids of
 appropriately prepared mice has been measured by adapting methods used for the bioassay of
 thyrotropin. One minimum lethal dose of tetanus toxin given sc caused a significant release of
 radioiodine into the blood of mice 48 h after injection. In mice subjected to the stress of prior
 bleedings or anesthesia, the release of radioiodine from the thyroid by tetanus toxin was
 accelerated, i.e., the increase in blood radioiodine could be measured 24 h after injection. These
 results again suggest that tetanus toxin may interact with thyrotropin receptors on thyroid plasma
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 Activation of T lymphocyte subsets by synthetic TSH receptor peptides
 and recombinant glutamate decarboxylase in autoimmune thyroid
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 Thyroid-stimulating antibody activity between different immunoglobulin
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 Thyrotropin-releasing hormone has profound presynaptic action on
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 Characteristics and bioefficacy of monoclonal antgonadotropin
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7/7/12 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
 03639753 82119926 PMID: 7328074
 Thyroid function in tetanus.

Dastur FD; Shah J; Awatramani V; Shah JM; Pardiwalla BS; Nair KG; Mehta MN; Desai KB
 Journal of the Association of Physicians of India (INDIA) Jul 1981, 29 (7) p519-25, ISSN
 0004-5772 Journal Code: HG7 Languages: ENGLISH Document type: Journal Article
 Record type: Completed Record Date Created: 19820412

7/7/15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
 03389313 79127176 PMID: 217603

Tetanus toxin interactions with the thyroid: decreased toxin binding to membranes from a
 thyroid tumor with a thyrotropin receptor defect and in vivo stimulation of thyroid function.
 Habig WH; Grollman EF; Ledley FD; Meldolesi MF; Aloj SM; Hardegree MC; Kohn LD
 Endocrinology (UNITED STATES) Mar 1978, 102 (3) p844-51, ISSN 0013-7227 Journal
 Code: EGZ Languages: ENGLISH Document type: Journal Article Record type: Completed
 Normal rat thyroid membranes adsorb neurotoxicity when incubated with purified tetanus toxin.
 Membranes from a rat thyroid tumor with a thyrotropin receptor defect adsorb very little

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 Demonstration and characterization of partial glyceride specific lipases
 in pig thyroid plasma membranes. Nov 28 1980

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 Defective regulation of the immune response to tetanus toxoid in
 Hashimoto's disease. Jul 1984

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 Action of phospholipase C on the thyroid. Abolition of the response to thyroid-stimulating
 hormone.
 Maccchia V; Pastan I

Antigenic determinants on human choriogonadotropin alpha-subunit. II.
 Immunochemical mapping by a monoclonal antipeptide antibody. Jul 25
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 Interaction of fragments B and C of tetanus toxin with neural and
 thyroid membranes and with gangliosides. Jul 10 1980

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 Gangliosides, the thyrotropin receptor, and autoimmune thyroid disease.
 1984

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 Thyrotropin receptors and gangliosides. 1980

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 Thyroid function in tetanus. Jul 1981

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 Structure: function studies of receptors for thyrotropin and tetanus
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Effects of concanavalin A and neuraminidase on cyclic AMP levels
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 Action of phospholipase C on the thyroid. Abolition of the response to
 thyroid-stimulating hormone. Apr 25 1967

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 Role of lecithin in the mechanism of TSH action. Apr 1970

syndrome characterized as "thyroid storm" in patients with Graves' disease are discussed as they
 may relate to these observations. Record Date Created: 19790516

Tags: Animal
 Descriptors: Protirelin--metabolism--ME; *Receptors, Cell Surface--metabolism--ME; *Tetanus Toxin--
 metabolism--ME; *Thyroid Gland--metabolism--ME; *Thyroid Neoplasms--metabolism--ME; Cell
 Membrane--metabolism--ME; Kinetics; Rats
 CAS Registry No.: 0 (Receptors, Cell Surface); 0 (Tetanus Toxin); 24305-27-9 (Protirelin)

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Clostridium septicum infection of the thyroid gland.
 Warren CP; Mason BJ
 Postgraduate medical journal (ENGLAND) Sep 1970, 46 (539) p586-8, ISSN 0032-5473
 Journal Code: PFX
 Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date
 Created: 19710204
 Tags: Human; Male
 Descriptors: Clostridium Infections --complications--CO; *Thyroiditis-etiology--ET; Adenocarcinoma--
 complications--CO; Clostridium--isolation and purification--IP; Colonic Neoplasms--complications--CO; Middle
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 Tetanus toxin and thyrotropin interactions with rat brain membrane
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 Immunological reactivity of antibodies produced by Pr-beta-HCG-TT
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 Tetanus toxin interactions with thyroid plasma membranes.
 Implications for structure and function of tetanus toxin receptors and
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neurotoxicity when similarly evaluated. This inability of the tumor membranes to adsorb
 neurotoxicity is correlated with a defect in their ability to bind both 125I-labeled tetanus toxin and
 [125I]iodothyrotropin. The effect of tetanus toxin on the release of radioiodine from the thyroids of
 appropriately prepared mice has been measured by adapting methods used for the bioassay of
 thyrotropin. One minimum lethal dose of tetanus toxin given sc caused a significant release of
 radioiodine into the blood of mice 48 h after injection. In mice subjected to the stress of prior
 bleedings or anesthesia, the release of radioiodine from the thyroid by tetanus toxin was
 accelerated, i.e., the increase in blood radioiodine could be measured 24 h after injection. These
 results again suggest that tetanus toxin may interact with thyrotropin receptors on thyroid plasma
 membranes. The "sympathetic overactivity syndrome" seen in some patients with tetanus and the
 syndrome characterized as "thyroid storm" in patients with Graves' disease are discussed as they
 may relate to these observations. Record Date Created: 19790516

Tags: Animal
 Descriptors: Protirelin--metabolism--ME; *Receptors, Cell Surface--metabolism--ME; *Tetanus
 Toxin--metabolism--ME; *Thyroid Gland--metabolism--ME; *Thyroid Neoplasms--metabolism--ME; Cell
 Membrane--metabolism--ME; Kinetics; Rats
 CAS Registry No.: 0 (Receptors, Cell Surface); 0 (Tetanus Toxin); 24305-27-9 (Protirelin)

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 Effect of sphingomyelinase from Clostridium perfringens on the meta-
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 The purification and properties of a thyroid-stimulating factor isolated
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Journal of biological chemistry (UNITED STATES) Apr 25 1967, 242 (8) p1864-9, ISSN
 0021-9258 Journal Code: HIV
 Languages: ENGLISH Document type: Journal Article Record type: Completed Subfile:
 INDEX MEDICUS
 Tags: Animal; In Vitro

Descriptors: Phospholipases—pharmacology—PD; * Thyroid Gland—drug—DE; *Thyrotropin—pharmacology—PD; Acetylcholine—pharmacology—PD; Clostridium—enzyme—EN; Dogs; Edetic Acid—pharmacology—PD; Glucose—metabolism—ME; Neuraminidase—metabolism—ME
 CAS Registry No.: 50-99-7 (Glucose); 51-84-3 (Acetylcholine); 60-00-4 (Edetic Acid); 9002-71-5 (Thyrotropin)
 (Thyrotropin) Enzyme No.: EC 3.1.- (Phospholipases); EC 3.2.1.18 (Neuraminidase) Record Date Created: 19670706

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 Spectroscopic investigation of calcium binding sites in the neurotoxin vipoxin and its components—relation with the X-ray structure. Dec 2000

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 Modulation of phospholipase A2 activity generated by molecular evolution. Oct 30 1999

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 Specific physiological roles of cytosolic phospholipase A(2) as defined by gene knockouts. Oct 31 2000

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 Crystal structure of agkistrotoxin, a phospholipase A2-type presynaptic neurotoxin from *Agkistrodon halys pallas*. Sep 11 1998

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 Isolation, purification, crystallization and preliminary X-ray analysis of beta 1-bungarotoxin from *Bungarus caeruleus* (Indian common krait). May 1999

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 Postsynaptic alpha- neurotoxin gene of the spitting cobra, *Naja naja sputatrix*: structure, organization, and phylogenetic analysis. Mar 1999

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The purification and properties of id-stimulating factor isolated from *Clostridium perfringens*.

Macchia V; Bates RW; Pastan I
 Journal of biological chemistry (UNITED STATES) Aug 25 1967, 242 (16) p3726-30, ISSN 0021-9258 Journal Code: HIV
 Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19671022

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 A monoclonal antibody directed against the non-toxic subunit of a dimeric phospholipase A2 neurotoxin, crotoxin, neutralizes its toxicity. Jul 1998

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 Spectroscopic properties and stability of the neurotoxic complex. Vipoxin and its components. Aug 1998

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 The histamine releasers croptamine, protamine and compound 48/80 activate specific proteases and phospholipases A2. Sep 1997

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Effect of phosphorylation of synapsin I and other synaptosomal proteins by beta-bungarotoxin, a phospholipase A2 neurotoxin. Dec 1992
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Crotoxin, half-century of investigations on a phospholipase A2 neurotoxin. 1989
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Crotoxin, a phospholipase A2 neurotoxin from the South American rattlesnake *Crotalus durissus terrificus*: purification of several isoforms and comparison of their molecular structure and of their biological activities. Jan 26 1988
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Studies on the subunit structure of textilotoxin, a potent neurotoxin from the venom of the Australian common brown snake (*Pseudonaja textilis*). Sep 24 1987
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Binding ability of *Clostridium botulinum* neurotoxin to the synaptosome upon treatment of various kinds of the enzymes. Mar 30 1987
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Characterization of monoclonal antibodies against beta-bungarotoxin and their use as structural probes for related phospholipase A2 enzymes and presynaptic phospholipase neurotoxins. Jul 2 1984
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Sequence homology between phospholipase and its inhibitor in snake venom. The primary structure of the inhibitor of vipoxin from the venom of the Bulgarian viper (*Vipera ammodytes ammodytes*, Serpentes). Aug 1984
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Isolation of "ceruleotoxin" from *Bungarus fasciatus* venoms. 1982
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Synergism of the two subunits of crotoxin. 1982
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Amino acid sequence of phospholipase A from *Bungarus multicinctus* venom. Jan 1981
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[Release of 14C-acetylcholine from synaptosomes as affected by presynaptic neurotoxins from bee and cobra venoms—phospholipases A2] *Vysvobozhdenie [14C] atsetilkholina iz sinaptosom pod vlianiem presinapticheskikh neirotoksinov iz iadov zmei i pcheley—fosfolipaz A2*. Nov 1983
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Penetrability of proteins through the digestive system of *Sarcophaga falcata* blowfly. Jan 3 1980

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Neuronal degeneration induced by stereotaxic injection of beta-bungarotoxin into rat brain. Feb 1979

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Relationship between the neurotoxicity and phospholipase A activity of beta-bungarotoxin. Jan 11 1977

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Saturable binding to cell membranes of the presynaptic neurotoxin, beta-bungarotoxin. May 21 1976

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The role of phospholipase activity in the action of a presynaptic neurotoxin from the venom of *Notechis scutatus scutatus* (Australian tiger snake). Jan 1 1976

11/7/146 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
08140009 94223544 PMID: 8169833

Inhibition of vacuolar adenosine triphosphatase antagonizes the effects of clostridial neurotoxins but not phospholipase A2 neurotoxins.

Simpson LL; Coffield JA; Bakry N
Department of Medicine, Jefferson Medical College, Philadelphia, Pennsylvania.
Journal of pharmacology and experimental therapeutics (UNITED STATES) Apr 1994, 269 (1) p256-62, ISSN 0022-3565 Journal Code: JP3 Contract/Grant No.: NS-22153, NS, NINDS Languages: ENGLISH Document type: Journal Article Record type: Completed
Bafilomycin A1, an inhibitor of vacuolar adenosine triphosphatase, was tested for its ability to — antagonize botulinum neurotoxins (serotypes A-G), tetanus toxin and phospholipase A2 neurotoxins (notexin, beta-bungarotoxin, taipoxin and textilotoxin) on the mouse phrenic nerve-hemidiaphragm preparation. Bafilomycin itself produced concentration-dependent blockade of neuromuscular transmission without blocking nerve action potentials or muscle action potentials. This effect may have been due to inhibition of the proton pump that regulates acetylcholine transport into vesicles. At submaximal concentrations, bafilomycin was very effective in delaying the onset of paralysis due to all clostridial neurotoxins, but it had no protective effect against phospholipase A2 neurotoxins. Experiments were done to determine which of the three steps in clostridial neurotoxin action was antagonized by bafilomycin (e.g., binding, internalization and intracellular poisoning). Both pharmacological experiments and ligand-binding experiments showed that the drug did not block toxin binding to the plasma membrane. Similarly, pharmacological experiments on the time-dependent effects of bafilomycin showed that the drug did not antagonize the intracellular actions of toxins. The data indicated that bafilomycin acted at the intermediate step of internalization. This is in keeping with the facts that: 1) bafilomycin inhibits vacuolar adenosine triphosphatase, which in turn leads to inhibition of acidification in endosomes and 2) clostridial neurotoxins depend upon acidification of endosomes for translocation to the cytosol. The finding that bafilomycin antagonizes tetanus toxin may provide important clues for understanding how this toxin can act locally to produce flaccid paralysis. The finding that bafilomycin is a universal antagonist that protects against all clostridial neurotoxins may have important implications for developing therapeutic drugs. Record Date Created: 19940602

11/5/49 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
08081260 94066007 PMID: 8246147

Chelation of zinc antagonizes the neuromuscular blocking properties of the seven serotypes of botulinum neurotoxin as well as tetanus toxin.

Simpson LL; Coffield JA; Bakry N
Department of Medicine, Jefferson Medical College, Philadelphia, Pennsylvania.
Journal of pharmacology and experimental therapeutics (UNITED STATES) Nov 1993, 267 (2) p720-7, ISSN 0022-3565 Journal Code: JP3 Contract/Grant No.: NINCDS NS-22153, NS, NINDS Languages: ENGLISH Document type: Journal Article Record type: Completed
Botulinum neurotoxin types A, B (unactivated and activated), C, D, E, F and G, as well as tetanus toxin, paralyzed transmission in mouse phrenic nerve-hemidiaphragm preparations. Toxin-induced blockade of transmission was antagonized by chelators [e.g., ethylenediamine tetraacetic acid, tetrakis(2-pyridylmethyl)ethylenediamine or diethylene-triaminepentaacetic anhydride], but
17/6/1 10817918 20332947 PMID: 10874773
Ocular aspects of myasthenia gravis. 2000

17/6/2 10358931 20000569 PMID: 10532769
Strabismus surgery among aged medicare beneficiaries. Dec 1997

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The posterior thyroplasty window: anatomical considerations. Aug 1999

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Botulinum toxin A treatment of overactive corrugator supercilii in thyroid eye disease. May 1998

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Control of eyelid retraction associated with Graves' disease with botulinum A toxin. Mar 1994

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Amino acid sequence of a presynaptic neurotoxin from the venom of *Notechis scutatus scutatus* (Australian tiger snake). Sep 10 1975

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Snake venom action: are enzymes involved in it? Feb 15 1977

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Effect of presynaptic neurotoxin notechis II-5 from tiger snake venom on the motor nerve endings of mice] Deistvie presinaphticheskogo neirotoksina notechisa II-5 iz iada tigrovoi zmei na dvigatel'nye nervnye okonchaniya myshi. Oct 1979

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Postsynaptic effects of crotoxin and of its isolated subunits. Sep 1979

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Amino acid sequence of a postsynaptic neurotoxin from the venom of the Australian tiger snake *Notechis scutatus scutatus*. 1979

this effect was dependent on incubation conditions. Pretreatment of toxin with chelators failed to produce antagonism, but pretreatment of tissues did produce antagonism. Of the various chelators tested, tetrakis(2-pyridylmethyl)ethylenediamine produced the greatest effect. Antagonism of toxin-induced neuromuscular blockade could be partially reversed by washing chelators from tissues and could be fully reversed by adding an excess of zinc. The ability of chelators to antagonize clostridial neurotoxins was specific and did not extend to phospholipase A2 neurotoxins. Ligand-binding studies with radiolabeled toxin and brain membrane preparations showed that chelators did not antagonize toxicity by inhibiting toxin association with receptors. Similarly, pharmacological experiments with unlabeled toxin- and type-specific antibodies demonstrated that chelators did not act by blocking receptor-mediated internalization of toxin. The chelators appeared to exert their effects by antagonizing the intracellular actions of clostridial neurotoxins. Electrophysiological studies showed that chelators, at concentrations relevant to antagonism of botulinum neurotoxin and tetanus toxin, did not enhance transmitter release.(ABSTRACT TRUNCATED AT 250 WORDS) Record Date Created: 19940106

Tags: Animal; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Descriptors: *Botulinum Toxins--antagonists and inhibitors--AI; *Chelating Agents--pharmacology--PD; *Neuromuscular Blocking Agents--antagonists and inhibitors--AI; *Tetanus Toxin--antagonists and inhibitors--AI; *Zinc--metabolism--ME; Botulinum Toxins--classification--CL; Botulinum Toxins--metabolism--ME; Brain--metabolism--ME; Chelating Agents--metabolism--ME; Metalloendopeptidases--metabolism--ME; Mice; Neuromuscular Junction--drug effects--DE; Neuromuscular Junction--metabolism--ME; Rats; Sensitivity and Specificity; Serotyping; Tetanus Toxin--classification--CL; Tetanus Toxin--metabolism--ME CAS Registry No.: 0 (Botulinum Toxins); 0 (Chelating Agents); 0 (Neuromuscular Blocking Agents); 0 (Tetanus Toxin); 7440-66-6 (Zinc)
Enzyme No.: EC 3.4.24 (Metalloendopeptidases)

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05067511 87184609 PMID: 3566763

Binding ability of Clostridium botulinum neurotoxin to the synaptosome upon treatment of various kinds of the enzymes.

Kitamura M; Sone S
Biochemical and biophysical research communications (UNITED STATES) Mar 30 1987, 143 (3) p928-33, ISSN 0006-291X Journal Code: 9Y8 Languages: ENGLISH Document type: Journal Article Record type: Completed
The binding ability of Cl. botulinum neurotoxin to synaptosomes upon treatment with various enzymes (neuraminidase, trypsin, and beta-bungarotoxin containing phospholipase A2 activity) was studied. When synaptosomes were treated with neuraminidase, their ability to bind toxin decreased; trypsin and beta-bungarotoxin had slightly weak or no effect. The decrease in toxin-binding ability of synaptosomes was paralleled by a release of sialic acid from the synaptosomes by the neuraminidase treatment. The toxin-binding ability of synaptosomes treated with neuraminidase was lower than untreated ones at a high concentration of sodium chloride. The binding of the toxin to synaptosomes occurred at least at the two types of structural sites, one site which contained sialic acid, and other site which was sensitive to high ionic strength. It may be possible that another binding state except these is present at the synapse. Record Date Created: 19870518

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[Myasthenic syndromes linked to mediator secretion disorders] Miastenicheskie sindromy, svyazannyye s narusheniem sekretnii mediatora. 1985

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Botulinum toxin therapy of eye muscle disorders. Safety and effectiveness. American Academy of Ophthalmology. Sep 1989

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Diplopia in thyroid eye disease. 1986

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Saccadic velocity measurements in strabismus. 1983

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Injection treatment of endocrine orbital myopathy. Aug 15 1984

17/7/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.

10240117 99371424 PMID: 10443824

The posterior thyroplasty window: anatomical considerations.

Maragos NE

Mayo Clinic, Rochester, Minnesota 55905, USA.

Laryngoscope (UNITED STATES) Aug 1999, 109 (8) p1228-31, ISSN 0023-852X

Journal Code: L1W Languages: ENGLISH Document type: Journal Article Record type:

Completed

OBJECTIVES: Explain surgical technique of performing a posterior thyroplasty window. Describe the internal laryngeal anatomy and structures available through the posterior window approach.

Describe posterior window approach. STUDY DESIGN: Review of lateral laryngeal anatomy and retrospective review of 125 cases involving a posterior thyroplasty window approach. Review mechanics of stress and stress concentration inherent with partial removal of rigid substance.

Describe anatomical considerations and surgical complications. METHODS: Charts were reviewed and tabulated for surgical complications, efficacy and safety of surgical approach, specific anatomical variations, and variety of surgery available through the posterior window.

RESULTS: Performance of 125 posterior thyroplasty windows revealed no evidence of entry into the piriform sinus. Three thyroid ala fractures ensued, two of the body and one of the inferior cornu. Operations available included arytenoid adduction, arytenoid fixation, lysis of joint adhesions, and access to the posterior cricoarytenoid muscle for botulinum toxin injections.

CONCLUSIONS: The posterior thyroplasty window affords easy, direct access to the internal, posterolateral larynx while preserving the cricothyroid joint, the action of the cricothyroid muscle, and the internal division of the recurrent laryngeal nerve. Record Date Created: 19990907

1777/8 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 06895291 93064763 PMID: 1437204

Bilateral thyroarytenoid denervation: a new treatment for laryngeal hyperadduction disorders studied in the canine.

Sercarz JA; Berke GS; Ming Y; Rothschilder J; Graves MC

UCLA School of Medicine, Division of Head and Neck Surgery.

Otolaryngology-head and neck surgery (UNITED STATES) Nov 1992, 107 (5) p657-68,

ISSN 0194-5998 Journal Code: ON8 Languages: ENGLISH Document type: Journal Article

Record type: Completed

Adductor spasmodic dysphonia is a vocal disorder of uncertain etiology with no satisfactory long-term treatment. Recently, injection of botulinum toxin (Botox) into the thyroarytenoid (TA) muscle has been used as an effective temporary treatment. A surgical counterpart to bilateral TA Botox injection is described in this article. Bilateral thyroarytenoid denervation was performed through a window in the thyroid cartilage in seven canines, including four that were studied 3 months after the procedure. No serious complications occurred in the animals, each maintaining full vocal fold abduction and adduction. In all cases, anticipated physiologic changes in laryngeal function were observed, including the inability to generate high subglottic pressures during high levels of laryngeal nerve (RLN) stimulation. In two of the surviving animals, the ansa cervicalis was used to reinnervate the TA muscle, thereby preventing the possibility of reinnervation from the proximal RLN stump while limiting TA atrophy and fibrosis. Bilateral TA denervation represents a hopeful new long-term approach to spasmodic dysphonia treatment. Record Date Created: 19921218

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Botulinum toxin.

Kowal L

Australian and New Zealand journal of ophthalmology (AUSTRALIA) Aug 1988, 16 (3)

p264-6, ISSN 0814-9763 Journal Code: ANZ Languages: ENGLISH Document type: Journal

Article Record type: Completed Record Date Created: 19881220

1777/16 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 05131078 87070940 PMID: 3466462

Diplopia in thyroid eye disease.

Fells P; McCarty B

Transactions of the ophthalmological societies of the United Kingdom (ENGLAND) 1986,

105 (Pt 4) p413-23, ISSN 0078-5334 Journal Code: WA1 Languages: ENGLISH Document

type: Journal Article Record type: Completed Record Date Created: 19870122

S9 23407 NEUROTOX?

S10 0 S7 AND S9

S11 1540602 TREAT?

S12 321 S11 AND S7

02jul01 08:32:48 User208600 Session D1405.1

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Set	Items	Description	
S1	6430	BOTUL?	
S2	17401	CALCITONIN	
S3	8	S1 AND S2	
S4	19552	HYPOCALC? OR HYPERCALC?	
S5	5	S1 AND S4	
S6	90754	THYROID	
S7	1231	S4 AND S6	
S8	0	S1 AND S7	
	36/1	09948794 98433827 PMID: 9762863	
		Effects of botulinum neurotoxin type A on the expression of gephyrin in cat abducens motoneurons. Oct 12 1998	
	36/2	09471682 98014520 PMID: 9344559	
		Sensory and motor denervation influence epidermal thickness in rat foot glabrous skin. Oct 1997	
	36/3	09404508 97291189 PMID: 9145803	
		Expression of neurotransmitter genes in rat spinal motoneurons after chemodenervation with botulinum toxin. May 1997	
	36/4	09159891 97110858 PMID: 9081635	
		NSW Agriculture, Agricultural Research and Veterinary Centre.	
		Australian veterinary journal (AUSTRALIA) Jun 1995, 72 (6) p228-34, ISSN 0005-0423 Journal Code: 91E	
		09088502 96364144 PMID: 8739382	
		Upregulation of calcitonin gene-related peptide at mouse motor nerve terminals poisoned with botulinum type-A toxin. 1996	
		Meunier FA; Colasante C; Faillie L; Gastard M; Molgo J	
		Laboratoire de Neurobiologie Cellulaire et Molculaire, C.N.R.S. (U.P.R.9040), Gif-sur-Yvette, France.	
		Plügers Archiv (GERMANY) 1996, 431 (6 Suppl 2) pR297-8, ISSN 0031-6768 Journal Code: OZX	
		Calcitonin gene-related peptide (CGRP)-like immunoreactivity of motor nerve terminals was investigated at different times after local in vivo injection of botulinum type-A toxin (BoNT/A) close to the mouse levator auris longus muscle. CGRP expression in most of control nerve terminals was undetectable, but markedly increased during muscle paralysis and synaptic remodelling and declined once functional recovery occurred. Record Date Created: 19970116	
		57/1/1 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.	
		10922488 20471417 PMID: 11021434	
		Fatal Clostridium botulinum intoxicosis in eleven Holstein cattle fed round bale barley haylage.	
		Kelch WJ; Kerr LA; Pringle JK; Rohrbach BW; Whitlock RH	
		Department of Comparative Medicine, College of Veterinary Medicine, University of Tennessee, Knoxville 37901-1071, USA.	
		Journal of veterinary diagnostic investigation (UNITED STATES) Sep 2000, 12 (5) p453-5, ISSN 1040-6387 Journal Code: AZD Languages: ENGLISH Document type: Journal Article Record type: Completed	
		Twenty-two lactating Holstein cattle in Tennessee had clinical signs of intoxication with preformed Clostridium botulinum toxin. These signs included weakness, paralysis of the tongue and chest muscles, abdominal breathing, and, in 11 of the 22 cows, death. Differential diagnoses included hypocalcemia, hypomagnesemia, carbohydrate overload, and several toxicoses including mycotoxin, lead, nitrate, organophosphate, atropine or atropine-like alkaloid, and botulism. A diagnosis of botulism by the ingestion of preformed C. botulinum type B toxin was made by eliminating these other diseases, by finding C. botulinum type B spores in 3 bales of round bale barley haylage fed to these cattle, and by isolating preformed type B toxin from 1 of the 3 bales. Confirmation of the toxin type was made by demonstrating mouse lethality by intraperitoneal injection of specimen extracts with neutralization by C. botulinum type B antitoxin. The haylage, harvested green and encased in black plastic bags to facilitate fermentation, was presumably contaminated by the botulinum toxin when fermentation failed to produce enough acid to lower the pH to 4.5. The pH below which C. botulinum growth is inhibited. Farmers and ranchers who use round hay bales to produce haylage should be alert to this potential problem. Record Date Created: 20010108	
		57/2/2 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.	
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		The clinical differentiation of nervous and muscular locomotor disorders of sheep in Australia.	
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		56/4 06402894 87186703 PMID: 3494493	
		Localized diseases of the bovine brain and spinal cord. Mar 1987	
		56/5 03657791 83094030 PMID: 7348919	
		[Central nervous system diseases in cattle. 2. Diseases in young and adult cattle] Erkrankungen des Zentralnervensystems beim Rind. 2. Die Krankheiten jugendlicher und erwachsener Rinder. 1981	
		126/1 11321719 21280324 PMID: 11386024	
		Coexistence of parathyroid carcinoma and non-medullary carcinoma of the thyroid. May-Jun 2001	
		126/2 11317161 21270595 PMID: 11376402	
		Minimally invasive video-assisted parathyroidectomy: multinstitutional study. Jun 2001	
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		Short- and long-term results of total vs subtotal thyroidectomies in the surgical treatment of Graves' disease. 2001	
		126/4 11303377 21132285 PMID: 11236346	
		Incidence of complications of thyroid surgery. Incidenza delle complicanze nella chirurgia della tiroide. Oct 2000	
		126/5 11256203 21241882 PMID: 11344634	
		Regulation of motoneuronal calcitonin gene-related peptide (CGRP) during axonal growth and neuromuscular synaptic plasticity induced by botulinum toxin in rats. Apr 1996	
		36/5 09088502 96364144 PMID: 8739382	
		Upregulation of calcitonin gene-related peptide at mouse motor nerve terminals poisoned with botulinum type-A toxin. 1996	
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		Calcitonin gene-related peptide: possible role in formation and maintenance of neuromuscular junctions. Jan 1995	
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		Calcitonin gene-related peptide-like immunoreactivity, in botulinum toxin-paralysed rat muscles. Sep-Nov 1994	
		36/8 07960661 94061378 PMID: 8242236	
		Enhancement by calcitonin gene-related peptide of nicotinic receptor-operated noncontractile Ca2+ mobilization at the mouse neuromuscular junction. Oct 1993	
		Many of the nervous and muscular locomotor disorders that affect sheep throughout Australia are commonly referred to as "stagers" syndromes. The range of clinical signs displayed by sheep suffering these disorders is sufficiently diverse to enable each syndrome to be graded into one of 5 progressive clinical groups. The first group, the limb paresis syndromes, includes primary myopathies associated with the ingestion of Ixolaena brevicompta, Malva parviflora, and Trachymene ochracea, as well as selenium and Vitamin E disorders. Parvo virus staggers, congenital progressive muscular dystrophy, humpy back, hypocalcaemic muscle weakness, Tribulus terrestris staggers and tetanus. The second group is characterised by limb paresis with knuckling of the fetlocks, and includes the plant-associated toxicities of Romulea rosea, Stachys arvensis, Trachyantha divaricata, and Tribulus micrococcus, together with haloxon toxicity, enzootic ataxia (copper deficiency), and the probably genetic disorders of segmental axonopathy, neuroaxonal dystrophy, and degenerative thoracic myelopathy. Other locomotor disorders that fit more loosely into this group are listerial myelitis (post-dipping staggers), vitamin A deficiency, cervico-thoracic vertebral subluxation Stypandra glauca toxicity, Ipomoea spp toxicity, ivermectin toxicity, and botulism. The third group, the falling syndromes, includes the probably genetic disorders of thalamic cerebellar neuropathy, cerebellar atrophy, and globoid cell leukodystrophy, together with Swainsona spp toxicity. The fourth group, the falling syndromes, includes the plant associated toxicities of phalaris staggers, perennial ryegrass staggers and nervous ergotism (Claviceps paspali). (ABSTRACT TRUNCATED AT 250 WORDS) (40 Refs.) Record Date Created: 19960122	
		57/3/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.	
		07689557 93149376 PMID: 1491765	
		[Primary hyperparathyroidism with prevalent neuro-muscular manifestations] [periparatiroidismo primario con prevalenti manifestazioni neuro-muscolari.	
		Bartolucci L; Fioretti M; Fioroni G; Proietti MG; Gradoli C; Valori C	
		Istituto di Patologia Speciale Medica, Università degli Studi di Perugia, Ospedale Civile, Terni.	
		Minerva medica (ITALY) Dec 1992, 83 (12) p841-5, ISSN 0026-4806 Journal Code: N6M	
		Minerva medica (ITALY) Document type: Journal Article Record type: Completed	
		A case of primary hyperparathyroidism with prevalent neuromuscular symptoms is described. Clinical, diagnostic and therapeutic implications are emphasized. Particular attention must involve a full clinical examination, electromyographic data, neuromuscular biopsy to make differentiation from primary myopathy or denervation pathology. Some similarity of electromyographic data with those observed in botulism and myasthenia gravis should also be taken in mind. Hypercalcaemia could play a pathological role in conditioning abnormalities of nervous impulse conduction at the level of neuromuscular junction. Another possible interference might be related to a direct effect of parathormone and hypophosphataemia on nervous impulse conduction. "Glandular hyperplasia", as observed in this case at histologic examination, rises some problems as far as the prognosis is concerned. Record Date Created: 19930304	
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		Review of the outcome of management of Graves' disease in children and adolescents. Apr 2001	
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126/12 11070816 21129969 PMID: 11234312	Short- and long-term results of total vs subtotal thyroidectomies in the surgical treatment of Graves disease. 2001	126/12 11070816 21129969 PMID: 11234312	Change of surgical treatment in benign thyroid gland diseases-- influence of surgical management on perioperative complications] Wandel der chirurgischen Therapie bei benignen Schilddrüsenerkrankungen-- Einfluss der chirurgischen Taktik auf perioperative Komplikationen. 2000	126/49 10171189 99283054 PMID: 10354657	126/47 10237517 99241664 PMID: 10225153
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